pp. 877-954] [December, 1943

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MAIN CONTENTS :

	PAGES		PAGES
Malaria	877-883	Relapsing Fever and other Spirochaetoses	916-919
Blackwater Fever	883-885	Yaws	919-921

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BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

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MALARIA.

COGGESHALL, L. T. **Malaria as a World Menace.** *J. Amer. Med. Ass.* 1943, May 1, v. 122, No. 1, 8–11.

A very large proportion of the American Army is serving in parts of the world in which malaria is hyperendemic. The author paints a very gloomy picture of the possible consequences not only to the soldiers themselves but also to their homes, to which they will return bringing new strains of plasmodia with them. "The potential danger of malaria during the present war is a greater worldwide menace than ever before." [Such forebodings are natural enough to all who have worked in malarious countries, but as there appears to be so little that can be done to falsify them it is of doubtful wisdom to give them undue publicity.] *Norman White.*

ZOZAYA, C. Paludismo y arrozales. [**Malaria and Rice Fields.**] *Rev. Facul. de Méd.* Bogotá. 1943, Feb., v. 11, No. 8, 448–76.

ROSS, E. S. & ROBERTS, H. R. **Mosquito Atlas. Part I. The Nearctic Anopheles, Important Malaria Vectors of the Americas and** *Aëdes aegypti*, *Culex quinquefasciatus*. pp. I. iv+I. 44, numerous figs. (1 map). 1943, May 28. Philadelphia: American Entomological Society, 1900 Race Street. [$·60.]

The authors' idea is original. The "atlas" consists of a number of full page text figures of larvae and adult anophelines. For each species the same set of parts is illustrated, *e.g.*, for adults, wing, mouthparts, hind tarsus, mesonotum, male terminalia, phallosome, and claspers. There are a few extra plates, giving a map of the races of *A. maculipennis* in N. America, two domestic culicines and *A. gambiae* (which might again claim citizenship in the New World). The work is produced as a pamphlet, but all pages are pierced so that they could be separated and used in a loose leaf cover. This would enable other species to be incorporated [a point of importance, for the authors have limited themselves to "important malaria vectors," but the malariologist often wants to know the other species sufficiently well to exclude them].

The figures are line drawings and appear to be extremely good: magnifications should be given. At the bottom of each plate are

brief notes (a dozen lines in all), on identification, adult habits, synonyms, larval habitat, distribution, and source of actual material on which the figure is based.

There is introductory information on the anatomy of adult and larva, and methods of mounting. Compressed notes on field methods would be acceptable.

Further parts dealing with the anophelines of the Old World are in preparation. *P. A. Buxton.*

KUMM, H. W., BUSTAMANTE, M. E. & HERRERA, J. R. Informe sobre algunos Anopheles hallados en la frontera de Mexico y Guatemala. (1942). [**Certain Anopheles found on the Borders of Mexico and Guatemala.**] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1943, June, v. 4, No. 2, 183–7, 1 map. [11 refs.]

LINDSAY, D. K. **Guidance Notes on Pernicious Malaria.** [Correspondence.] *Trans. Roy. Soc. Trop. Med. & Hyg.* 1943, July, v. 37, No. 1, 63–6.

The author summarizes conclusions reached in dealing with pernicious malaria in unusual conditions. A valley noted for the malignancy of its fever, whose population had a hundred per cent. spleen rate, suddenly became the temporary home of 30,000 non-immune workers of a dozen races. The annual fever incidence rate of these susceptible foreigners was 250 per cent. and the type of malaria was unusually virulent. These notes on pernicious malaria as it occurred there were put together for the guidance of medical officers who came from time to time to help.

Pernicious malaria is an acute emergency in which even minutes may count. The four main types of pernicious malaria met with were algid, cerebral, haemorrhagic and gastro-intestinal. In the algid type the patient is found cold, pulseless and often unconscious. Treatment must be immediate, hot-water bottles, stimulant injections, one pint of intravenous saline containing six grains of quinine bihydrochloride. If open operation is needed a vein on the front of the ankle is recommended. The cerebral type of pernicious malaria was of two varieties, quiet and rowdy. In the quiet variety the patient may be only drowsy or in deep coma and usually has a temperature of 103° or 104°F. There is often incontinence of urine and faeces. Treatment is by lumbar puncture (partly to exclude meningococcal infection) and intravenous injection of quinine, 6 grains in 10 cc. of water, repeated every eight hours till drugs can be retained by mouth. In the rowdy variety the patient may be restless, semi-conscious, with noisy breathing and frothing at the mouth, or violent. The violent patient may be fighting mad, hard to catch and need eight men to hold him down. Morphia is indicated and chloroform may be needed before treatment can be given. These patients should be nursed on the floor. Lumbar puncture and intravenous quinine are indicated. In the violent patient mental stability may be regained in 6 to 12 hours; in the "restless" patient recovery is slower, and fatal results are common. The haemorrhagic type of pernicious malaria may be characterized by epistaxis, rectal haemorrhage, or petechial haemorrhages. Treatment is by intravenous quinine followed by antimalarial drugs by mouth. In the gastro-intestinal type there may be persistent vomiting, persistent hiccough, intestinal colic or choleraic symptoms. In this type also

intravenous quinine is indicated until drugs can be retained by mouth. Morphia and chloroform are sometimes useful in hiccough cases.

"The microscope has little place in the diagnosis of pernicious malaria. A negative blood-slide has sent many to the grave." Malaria may be responsible for some of the symptoms in cases of cerebrospinal meningitis or in cases of head injury. In that valley every person brought in unconscious should be given intravenous quinine no matter what other treatment may be needed. In cerebral cases quinine may be given intrathecally if lumbar puncture be done, but the procedure does not appear to have any therapeutic advantages. Quinine intramuscularly is absorbed more slowly than when given by mouth and has no place in the treatment of pernicious malaria except in young children. Intramuscular quinine, however, was popular; some 2,000 intramuscular injections were given in the "market-place"; only one abscess was recorded. After being got under control malaria may show an inversion of temperature; thus a patient who has had his fever at a stated hour may exhibit a decided drop in temperature at the same hour on the two days following the cessation of the attack. The classical sweating stage of malaria with its attendant weakness may be due more to the treatment, blankets during the shivering stage, than to the disease itself. In the treatment of malaria opium by mouth is useful but it is inferior to quinine.

[It is to be hoped that the author will deal at greater length on some future occasion with his experiences in the valley where malaria is such a killing disease.] *Norman White.*

TANGANYIKA TERRITORY. MEDICAL DEPARTMENT. Medical Pamphlet No. 37 (1942) (Replacing No. 13 of 1935). 6 pp. **The Treatment of Malaria.**

This pamphlet has been prepared for the information of medical practitioners in the Tanganyika Territory and summarizes recent trends in the specific treatment of malaria. It contains nothing at variance with accepted practice. Attention is called to the disadvantage of overtreating clinical malaria in partially immune persons such as compose the majority of the African population of the Territory; 40 grains of quinine or totaquina are generally sufficient to clear up an attack of malaria in such people. *Norman White.*

SEELER, A. O., DUSENBERY, E. & MALANGA, C. **The Comparative Activity of Quinine, Quinidine, Cinchonine, Cinchonidine and Quinoidine against** *Plasmodium lophurae* **Infection of Pekin Ducklings.** *J. Pharm. & Exper. Therap.* 1943, June, v. 78, No. 2, 159–63.

The Malaria Commission, League of Nations [this *Bulletin*, 1932, v. 29, 461] recommended that totaquine should contain not less than 15 per cent. quinine and at least 70 per cent. of crystalline cinchona alkaloids. The composition of totaquine is going to be re-defined in a supplement to the U.S.P. XII. The mixture will then contain not less than 7 per cent. or more than 12 per cent. of anhydrous quinine and a total of not less than 70 per cent. or more than 80 per cent. of anhydrous crystalline cinchona alkaloids [see this *Bulletin*, 1943, v. 40, 290]. In view of the altered composition of totaquine the antimalarial activity

of the four crystalline cinchona alkaloids and of two samples of totaquine and one of quinoidine has been determined in order to decide the effect of lowered quinine content. Their toxicity for mice was also investigated.

Numerous authors have reported on the activity of the above-mentioned substances in canary malaria. In the present case tests were made against the schizonts of *P. lophurae* in infections of ducks. Similar investigations of the action of drugs against this parasite have recently been made by WALKER and VAN DYKE [this *Bulletin*, 1942, v. 39, 441] and by MARSHALL, LITCHFIELD and WHITE [this *Bulletin* 1943, v. 40, 223]. Constant results are obtained if experimental conditions are uniform and the parasite responds to drugs which are effective in human malaria.

Peking ducklings about 50 gm. in weight were used and the infected blood was inoculated intravenously. The drugs were administered in water daily by a stomach tube, for the first six days after inoculation, in amounts proportional to body weight. In the toxicity experiments a single dose was likewise administered to mice. The fact that little difference was found in antimalarial activity or toxicity of the four crystalline alkaloids under the conditions of test suggests that a mixture of alkaloids should prove satisfactory as an antimalarial.

The following summary is given by the author :—

"1. Quinine, quinidine, cinchonine and cinchonidine show about the same activity against the schizonts of *P. lophurae* in Pekin ducklings.

"2. Quinoidine is considerably less active than the four crystalline cinchona alkaloids.

"3. Two samples of totaquine varying widely in their quinine content were as active as quinine in duck malaria.

"4. There is little difference in the acute oral toxicity for mice among the four crystalline alkaloids. Quinoidine is more toxic than the crystalline alkaloids." *J. D. Fulton.*

TOMMASO, P. Del grado di resistenza alla chinina di alcuni ceppi di "Plasmodium falciparum." [**Resistance to Quinine of certain Strains of** *P. falciparum.*] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1942, v. 1, No. 6, 77–84. English summary.

Notes are given of five cases of malaria, four *P. falciparum* infections and one with a mixed *P. falciparum* and *P. vivax* infection, all of which displayed an unusual resistance to quinine. All the infections had been contracted in the lowlands of Italian East Africa. The author puts forward the hypothesis that in areas such as these, where the sanitary level is low and the diet very deficient, *P. falciparum* may exhibit a more marked tendency to develop in tissue cells than do European strains, and thus be less exposed to the action of antimalarial drugs. *Norman White.*

GLAZKO, A. J. **Estimation of Quinine in Urine, including a Field Test for establishing the Ingestion of Quinine in Malarial Prophylaxis.** *U.S. Nav. Med. Bull.* 1943, Mar., v. 41, No. 2, 529–33, 1 fig. [11 refs.]

A method of detecting quinine in urine, using the double iodide of mercury (HgI_4K_2) as the precipitating agent, is described. The double

iodide is prepared by adding an excess of mercury, 30 gm., to a solution of 22 gm. of iodine and 30 gm. of potassium iodide in 20 cc. of distilled water. The mixture is shaken vigorously until most of the brown colour has disappeared, and then, while still yellow, the solution is cooled under a running tap. When cool it should be a light green colour. It is diluted to 400 cc. with distilled water and then decanted from the residual mercury. This solution will keep indefinitely. The sulphuric acid is made up to one part of the chemically pure acid in three parts of water. The combined reagent consists of equal volumes of the acid and double iodide solutions: this should be prepared fresh each day. To 5 cc. of urine in a test tube 5 cc. of the reagent are added. A milky opalescence indicates the presence of quinine or certain other cinchona alkaloids. Turbidity standards can be set up as an index of the quinine concentration. For field purposes a faint turbidity indicates that the man in question did not take his quinine the previous night. The presence of more than 5 mgm. per cent. albumin will give a flocculent precipitate with the reagent. The quinine iodomercurate dissolves on boiling, the albumin precipitate remains unchanged.

The method may be used for the quantitative estimation of quinine in urine, opalescence being measured with a photonephelometer. The technique is described in detail.

A number of experiments were carried out to determine the degree of turbidity that may be expected with urine collected at different intervals after the ingestion of quinine. A prophylactic dose of quinine taken at the evening meal gives a heavy turbidity with the reagent in urine collected the following morning. *Norman White.*

YOUNG, M. D., MCKENDON, S. B. & SMARR, R. G. **The Selective Action of Thiobismol on Induced Malaria.** *J. Amer. Med. Ass.* 1943, June 19, v. 122, No. 8, 492–4, 1 chart.

This is the record of an investigation to determine at what stage of development malaria parasites are affected by thiobismol, and consequently the optimum time of administration, and whether species of parasite other than *P. vivax* are affected by the drug. Observations on 13 white patients undergoing malaria therapy with *P. vivax* showed that a 0·1 or 0·2 gm. dose of thiobismol had an inhibitory effect on half-grown parasites, *i.e.* on parasites 16 to 28 hours after the last paroxysm that they had caused. Parasites older and younger than this did not seem to be affected. Within four hours of the intramuscular administration of the drug fragmentary and irregularly staining parasites were seen, as well as parasites free from red cells. By giving the drug 16 to 28 hours after a paroxysm that series of paroxysms was eliminated; a quotidian periodicity was changed to tertian and usually remained so throughout the remainder of the infection and sometimes through several subinoculations. Observations on nine negroes undergoing *P. malariae* therapy yielded no consistent results. Usually the parasite density decreased for several days after administration of thiobismol but the drug had no clear-cut action against any developmental stage of the quartan parasite. Thiobismol appeared to have no action at all on the parasites or fever of nine negroes undergoing malaria therapy with *P. falciparum.*

For the termination of *P. vivax* infections an injection of thiobismol the day quinine is started subdues fever more promptly than does quinine alone. *Norman White.*

RUSSELL, P. F. **Military Malaria Control.** *War Medicine.* Chicago. 1934, June, v. 3, No. 6, 565–84. [Refs. in footnotes.]

Practically the whole subject of malaria control is most ably condensed in this paper. The adjective "military" in the title of such a complete discussion is almost redundant: the principles of malaria prevention remain the same whatever population is at risk. The justification for its inclusion may perhaps be found in the somewhat greater attention paid to those control measures which the exigencies of military service in hyperendemic tropical countries may render most feasible. It is not possible to make a summary of such a vast amount of information nor is this necessary as much of that information is familiar. Attention may, however, be directed to a few points of special interest.

Spray-killing adult mosquitoes is a measure that is discussed at length. Among the preparations issued by the Quartermaster's Department of the United States Army is Freon-Pyrethrum Aerosol. This is a mixture of liquid freon 12 with 0·8 per cent. wax-free pyrethrins and 6·0 per cent. sesame oil. Freon 12 is dichlorodifluoromethane; its vapour pressure produces the necessary spraying pressure which does not decrease as long as a single drop of liquid is present in the closed container. The mixture is issued in pressure cans or cylinders of 18 fluid ounces capacity. As the freon 12 containing the insecticide is sprayed it forms a fine mist from which the solvent evaporates almost immediately leaving the pyrethrum and sesame oil as a cloud of fine droplets. Freon 12 is not toxic either to man or mosquito and it is not inflammable. Eighteen ounces of the mixture are sufficient to spray 150,000 cubic feet and are liberated in from 12 to 14 minutes of continuous use. No direct hits on mosquitoes should be attempted; this wastes spray. Freon-pyrethrum aerosol should make a valuable contribution to malaria control in the field.

Early in the paper it is emphasized that malaria can be controlled effectively only by specially trained men working in a co-ordinated and supervised organization. This seemed tantamount to a confession that in many circumstances arising in war in hyperendemic areas nothing effective could be done. This pessimistic outlook is corrected later when the importance of instructing all ranks in regard to malaria and malaria control receives recognition. Measures are described for which the individual soldier should make himself responsible, or in the carrying out of which he should give his intelligent cooperation, bed nets, protective clothing, sprays and repellents, with the use of suppressive antimalaria drugs. *Norman White.*

See also p. 943, WAR MEDICINE, Chicago, **Development of a Medical Service for Airline Operations in Africa.**

SOAP. New York. 1942, Nov., v. 18, No. 11, 91, 92 and 93. **Insecticides for the Army . . . The Aerosol Insecticide Programme and its Effects on the Insecticide Industry now and after the War.**

The Department of Agriculture, the Army and a few private companies have evolved a new type of self-propelling insect spray for use, principally against mosquitoes and flies, by the Army in the tropics. This is the "Freon bomb" which looks like a small rounded drum. The standard army size is 5 inches long and 2½ inches in diameter with

siphon tube running down the middle of the inside and a nozzle and valve release almost flush with the surface of the top.

The bomb contains pyrethrum concentrate dissolved in dichlorifluoromethane, known commercially as Freon 12, and used in refrigerators. This substance (which is non-toxic and non-inflammable) has a low boiling point and is kept under a pressure of 85 lb. to the square inch in the bomb. On opening the nozzle the insecticide is blown out and forms a fine fog or aerosol which is projected 6–8 feet from the nozzle. From that point it floats about like smoke.

It is estimated that the aerosol cloud from one bomb is sufficient to kill all flies and mosquitoes in 100,000 cubic feet.

The pyrethrum used is 20 per cent. concentrate and it is dissolved to give 4 mgm. pyrethrins per gm. of solution. It must be carefully filtered to remove all materials which might clog the jet. Refined sesame oil is added at 10 mgm. per gm. solution to increase the insecticidal effect. One bomb contains the equivalent of 1 lb. of pyrethrum flowers.

The bomb is filled by evacuating with a water pump, admitting the required amounts of pyrethrum concentrate and sesame oil and then connecting to the Freon supply. By heating the latter or by cooling the bomb, the liquid can be run into the bomb. A small amount of wax-like substance in the pyrethrum extract is not soluble in the Freon; but this adheres to the side of the bomb and causes no difficulty.

The bomb appears to be safe, simple and effective for use in the tropics. It seems especially suitable for killing mosquitoes in aircraft. Its disadvantages are: (i) The container must be returned to base for refilling, or be discarded; (ii) Irresponsible operators are tempted to waste pyrethrum by over use. *J. R. Busvine.*

MANWELL, R. D. & HATHEWAY, A. E. **The Duck as a Host for the Avian Malarias.** *Amer. J. Hyg.* 1943, Mar., v. 37, No. 2, 153–5.

WOLFSON has shown that the duck is susceptible to four species of bird malarial parasite—*P. cathemerium, P. relictum, P. elongatum* and *P. lophurae.* With a view to discovering if ducks are susceptible to other bird malarial parasites the experiments recorded in this paper were carried out. It was found that the duck could be infected with *P. nucleophilum, P. relictum* var. *matutinum* and *P. circumflexum* but not with *P. hexamerium, P. oti, P. rouxi, P. vaughani* or three strains of *P. cathemerium.* As regards *P. lophurae,* now employed extensively in white Peking ducks for chemotherapeutic work, it was found that the Mallard duck, the Muscovy duck and the common turkey were very susceptible. Pigeons developed only a light infection but canaries were infected without difficulty. *C. M. Wenyon.*

BLACKWATER FEVER.

FOY, H. & KONDI, Athena. **Lyso-Lecithin Fragility in Blackwater Fever and Haemolytic Jaundice.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1943, July, v. 37, No. 1, 1–18, 7 charts. [62 refs.]

Erythrocytes from cases of blackwater fever show an increased fragility to lyso-lecithin but normal erythrocytes transfused into a

patient suffering from blackwater fever are destroyed as readily as the patient's own cells. The cells in blackwater fever, therefore, possess some abnormal feature but it may be due to changes in their environment. Spherocytosis occurs and is accompanied by decreases in erythrocyte diameter-thickness ratios, volumes and areas, the values being intermediate between those of haemolytic jaundice and the normal. It may be that the initial stage in the destruction of the cells is a change in permeability of cell membrane which allows haemoglobin to escape. The cells later become swollen, " transparent " and finally disappear, but are not broken up for some time, because on resuspending in saline the " ghosts " reappear ; this phenomenon occurs both in lyso-lecithin and in snake venom haemolysis. If the process be allowed to continue, the cells are finally disrupted and will not reappear on saline resuspension. In haemolytic jaundice there may be some defect in the erythrocyte as well as a " splenic " factor, because splenectomy fails to alter the abnormal fragility of the cells although it may stop the periodic haemolysis. It has been suggested that haemolysis in many haemolytic conditions may be due to the production of lyso-lecithin following stagnation and separation of cells and plasma in the enlarged spleen. It is possible that lyso-lecithin fragility is related to the action of this substance on the lipoids or lipo-protein complex in the red cell membrane. The amount of lipoid in the cell envelope is related to the dimensions of the cell, and the ability of the cell to increase its volume without rupture is related to the lipo-protein ratio in its membrane. Lyso-lecithin haemolysis may be due to an enzyme, lecithinase, acting on a substrate of red cells or plasma lecithin to produce lyso-lecithin or some allied phosphatide ; lecithinases have been shown to be present in many conditions where haemolytic reactions occur, *e.g.*, in favism, snake poisoning and in *Cl. welchii* filtrates, as well as in the spleen and peripheral blood. There are various lecithinases, and the degradation products of their actions on lecithin are not always the same. Snake venom lecithinase acting on lecithin produces lyso-lecithin but the latter injected into baboons in relatively huge amounts produces no haemolysis and only a slight increase in cell diameter, which is in sharp contrast to the haemolytic reaction which follows injection of the venom. This difference may be due to the fact that in the former case the lecithin in the cells and plasma is not disturbed, whereas in the latter procedure the lecithinase will split the lecithin of the cells and plasma and so disturb the intra-cellular lipo-protein complex. There is some evidence to suggest the macrophages of the spleen elaborate a proteolytic enzyme capable of destroying both red and white cells and that the production of this enzyme runs parallel with blood cell phagocytosis. The part played by the reticulo-endothelial system in haemolysis, however, is not clear and quantitative studies to show the precise proportion of red cells destroyed by this system are lacking. Even when grossly hypertrophied it seems unlikely that it can account for the sudden cell destruction that occurs in the conditions under consideration. *F. Murgatroyd.*

NELSON, A. J. M. **Blackwater Fever : Review of a Case.** *Med. J. Australia.* 1943, May 29, v. 1, No. 22, 491–3, 1 fig.

This is a description of a severe attack of blackwater fever in an Australian soldier serving in Papua. From March to November 1942 he had lived in an isolated part of the country where there was no medical

officer. He took quinine, 5 grains daily, until his first attack of "fever" in May, after which he took 10 grains of quinine daily. He treated himself for this attack by remaining in bed and taking 30 grains of quinine daily for three days. During the following months he had about five attacks of fever but was able to keep at work. He was transferred to the Moresby area at the beginning of November. On November 6th he felt feverish and took a large dose of quinine. Blackwater fever developed next day and on November 8th he was admitted to hospital. The attack lasted for seven days with a short remission on the 4th day. The erythrocyte count fell from 2,500,000 per cmm. on the day of admission to 1,500,000 on the 6th day in hospital. He was able to drink well and passed large amounts of brownish-black urine. It was noted that on admission there was exquisite tenderness in both loins and in the lower part of the abdomen; the spleen was palpable four fingers' breadth below the ribs and the liver was enlarged and tender. Thick blood films showed scanty subtertian rings. He was given alkalis by mouth, and the urine soon became alkaline. Atebrin 0·1 gm. thrice daily and later 0·6 gm. daily, was also given by mouth. Blood transfusions were given on the 2nd, 3rd, 5th, 6th, 7th and 8th days, amounting altogether to 5½ litres. Anahaemin (liver extract) was given intramuscularly on the 7th and 9th days. He began to recover on the 8th day and was well and active by the 12th day. He returned to Australia and reported that he had remained well. *J. F. Corson.*

TRYPANOSOMIASIS.

MACAULAY, J. W. **A Tsetse Fly and Trypanosomiasis Survey in Bechuanaland 1940–1942.** 68 pp., 2 maps (1 folding) & 60 figs. [38 refs.] 1942. [? Mafeking]. [Summary taken from *Rev. Applied Entom.* Ser. B. 1943, Aug., v. 31, Pt. 8, 147–8.

As a result of the discovery of four cases of sleeping sickness in 1938 in Ngamiland, Bechuanaland Protectorate, where trypanosomiasis of stock is frequent and *Glossina* is reoccupying territory that it lost in the rinderpest outbreak towards the end of the last century, it was decided to make a sleeping-sickness survey of the human population and a tsetse-fly and trypanosomiasis survey of the country and stock. This report deals in detail with the latter survey, which was made between 1st July 1940 and 31st December 1941. The history of trypanosomiasis in Ngamiland is very briefly reviewed, an account is given of the procedure followed in the survey, and the physical characters of the country and the vegetation, population and game are described. *G. morsitans*, Westw., was the only species of *Glossina* found, and *Trypanosoma congolense* is the only animal trypanosome present. There are 112,403 head of cattle in Ngamiland, of which 45,272 are in threatened areas. Trypanosomiasis occurs frequently in cattle, only rarely in horses, probably because they are herded with greater care, and very occasionally in goats and donkeys, which are normally kept on or just within the fringe of the fly belt. Sheep are not usually grazed on any threatened area. A scheme for the economic organisation of trypanosomiasis control is suggested. It consists in the compulsory

sale of clinically infected stock to a treatment centre for a price greater than the value of the hide, and subsequent treatment and marketing. The importance of mechanical transmission in areas free from *Glossina* was confirmed in 1940.

The account of the survey and discussion upon it include much local detail. Catches of *G. morsitans* were always highest where vegetation was thickest and nearly always higher in thicket with canopy than any other type of vegetation. Breeding took place in the densest forest fringes, on account of the danger of desiccation in the open, where semi-arid conditions prevail. Pupae were not concentrated. Careful study of aerial photographs was found to assist in the location of breeding areas. Destruction of these by cutting small trees and low branches and burning when they were dry was undertaken as routine work in two areas. Subsequent catches showed that clearing made conditions in the immediate neighbourhood quite unsuitable for the fly in one area, but the results in the other were confused; their possible significance is discussed. The fly population did not appear, in the period of the survey, to be related to rainfall, or to maximum, minimum or mean temperature. The flies were more attracted by a screen bait consisting of a blanket suspended from a pole at shoulder height than by donkey and still more by man alone. Catches in a "new collapsible" trap of hessian and gauze, previously used in East Africa, were very disappointing, and apparently not influenced by position, but the numbers taken were too small to be significant. A trap similar in shape to the Harris trap but smaller also failed. No flies were taken in traps during the dry season, when trapping appears to be useless. Harris traps caught very well when there were men in the vicinity, but not at all otherwise.

In the discussion and conclusions, it is stated that humidity from swamps and the vegetative fringes to swamp channels provide the necessary shelter for the fly, and game is becoming more and more abundant. Advance into new areas is the result of population pressure and requires the availability of shelter and food in the form of game, domestic stock or, failing these, man. For advance to be permanent, there must also be suitable breeding sites, providing soil with a certain amount of humus and shelter from direct sun, wind and flood. Advance is halted by natural barriers consisting of areas that cannot support game or that lack shelter or in which game cannot be readily located, or by artificial barriers consisting of areas that have been invaded by fire or inhabited areas in which shelter and game are reduced. Local examples of the operation of these limiting factors are given, and "danger areas" into which the advance of the fly is considered possible unless the balance of factors is changed are discussed. These include Maun, the capital. The situation in the Chobe Fly Belt some 20 miles to the north, where there are 9,700 head of cattle, is briefly discussed. In the course of two visits, *G. morsitans* was the only species found. It is possible, but not proved, that the two belts are confluent.

Great persuasion will be required to bring the Ngamiland natives to support control work actively, and the administrative problems are discussed. Recommended measures include the selection of grazing grounds to be maintained, the settling of families along the edge of all danger areas, the erection of fences of thorn bush against game, thicket control, the provision of traps, the adoption of the scheme for compulsory treatment of infected stock, the control of game, and certain clearing work, details of which are given. No area should be allowed

to be a game reserve. References to *Glossina* in Ngamiland in works published between 1850 and 1917 are reviewed.

Since the conclusion of the sleeping-sickness survey, there has been a serious recrudescence in one area. A few notes on the occurrence of the disease and its dispersal are included, and the recommendations made by I. W. Mackichan in the report on the 1939–40 sleeping-sickness survey are given. They are that a sleeping-sickness patrol should be established, the inhabitants of certain villages transferred to tsetse-free areas, and return to the old villages or the erection of new ones in the fly-belt prohibited, and bush clearing in a specified area carried out.

Finally, it is suggested that Ngamiland could be surrounded by a cattle-free desert zone, all the cattle in Ngamiland and Chobe inoculated against rinderpest, and the disease then introduced among the large ungulates in the swamp area to reduce the food supply of the tsetse.

BRAZZAVILLE [AFRIQUE FRANÇAISE LIBRE]: RAPPORT SUR LE FONCTIONNEMENT TECHNIQUE DE L'INSTITUT PASTEUR EN 1941 [CECCALDI, J., Director]. 59–73. Contribution à l'étude de la réinfection en trypanosomiase humaine. [**Reinfection in Human Trypanosomiasis.**]

The African native often shows a surprising tolerance for human trypanosomes. A considerable number of people, apparently in perfect health, are found to be infected with trypanosomes on the occasion of a visit to the Pasteur Institute to obtain a health passport. In certain sleeping sickness patients the nervous stage does not develop for a very long time, sometimes years. In some cases also the disease may show periods of latency or " silence " lasting for years.

Notes are given of three patients who were treated and apparently cured, remaining well and showing no signs of infection on repeated examination during several years, and again found to be infected when examined in 1940-1941. Examinations were made two or three times a year by triple centrifugation of the blood and by lumbar puncture.

The first case was diagnosed and treated in 1929 and examined two or three times a year during 1930–1933 and then not seen again until 1941, when trypanosomes were found in his blood. The other two cases were diagnosed in 1932, treated and apparently cured. One was not seen after 1934 until December 1940, when trypanosomes were found in her blood; the other was not seen between June 1938 and May 1941, when the blood was negative by triple centrifugation but the cerebrospinal fluid showed trypanosomes, 588 cells per cmm. and 0·66 per cent. protein.

The question of persisting chronic or inapparent infection or of reinfection is discussed, but no conclusion is arrived at. *J. F. Corson.*

LEWIS, E. A., WILEY, A. J. & MACAULAY, J. W. **The Transmission of** *Trypanosoma congolense* **by** *Glossina austeni.* *Ann. Trop. Med. & Parasit.* 1943, Sept. 7, v. 37, No. 2, 98–107, 10 charts.

Pupae of *Glossina austeni* were collected near Kilifi in the Kenya coastal district, and the flies were bred out at the Veterinary Research Laboratory, Kabete, near Nairobi; transmission experiments with *Trypanosoma congolense* were made there. The strain of *T. congolense* was obtained from a naturally infected ox at Thika which is about 30 miles from Nairobi. Transmissions of *T. congolense* were made by

G. austeni and by inoculation of blood, to various animals including oxen, sheep, goats, pigs, mules, a dog, a buffalo, and an oryx. Observations were made of the incubation period and course of the disease by recording the body temperature and by microscopical examination of the blood. The infection was rapidly fatal to cattle, rabbits, mules and the dog, but was more chronic in sheep, goats, pigs and the oryx. The buffalo, which was infected by *G. austeni*, died on the 28th day but its death could not be attributed entirely to the trypanosome infection. The incubation period varied from about one to three weeks, occasionally longer.

Dissection of 21 of the flies showed infection in two; "the labrum contained dense colonies of the parasites, and the hypopharynx was lightly infested."

The authors conclude that it is reasonable to regard *G. austeni* as a natural vector of *T. congolense* and that additional experiments with "wild" flies are needed. *J. F. Corson.*

MAZZA, S. & CHACON, R. V. Primeros animales domésticos y seres humanos con *Schizotrypanum cruzi* comprobados en Bolivia. [**First Cases of *T. cruzi* Infection in Man and Domestic Animals of Bolivia.**] Reprinted from *Prensa Méd. Argentina.* 1943, Mar. 3, v. 30, No. 9, 8 pp., 2 figs.

Though infected Triatomid bugs have been known to occur in Bolivia for a long time, this was the only country in South America from which no human or other mammalian cases of infection with *Trypanosoma cruzi* had so far been recorded. In the present paper the authors report such cases for the first time. Working in different parts of Bolivia, and at various altitudes, they have examined thick blood films taken from 51 children, 15 pups and 4 kittens. Of these one child and one pup proved to be infected with the trypanosome. The authors further found a high degree of infection among Triatomid bugs (from 12·5 to 50 per cent. of those examined) caught in human dwellings. Inoculation of the faeces of these insects into white mice produced heavy infections. From these facts it is concluded that not only does Chagas's disease exist in Bolivia but its distribution in the country must be fairly wide. *C. A. Hoare.*

GASIC, G. Algunos hechos sobre clínica y epidemiologia de la enfermedad de Chagas en Chile. [**Facts regarding Chagas's Disease in Chile.**] *Bol. Oficina Sanitaria Panamericana.* 1943, Apr., v. 22, No. 4, 327–35. [14 refs.]

The study of Chagas's disease in Chile may be said to have started in 1937 when the Health Department set up a sub-department of Parasitology. The first acute case of the disease was recorded in 1938. Generally speaking, the disease appears to be of a milder type in Chile than in the Argentine, Brazil and Uruguay, and it is probable, nay certain, that many have passed unobserved, acute cases being exceptional and many of the more chronic being discovered by use of the Machado test on random persons among the population.

The author analyses 69 cases, of which 16 were acute, 48 subacute, and 5 chronic. Of the acute, 9 were children and 7 adults; 14 had palpebral oedema on one side, two had generalized oedema secondary to

the palpebral. Other signs were cardiac disturbances, mostly tachycardia, enlarged liver and spleen and generalized secondary glandular swelling. Two died with nervous and cardiac complications, the rest were relatively benign in course. The cardiac disturbances comprised changes in rate and rhythm, mostly of sinus origin. In 10 cases the trypanosome was found in the blood, in six proof was obtained by xenodiagnosis.

Subacute cases were characterized by tachycardia, enlargement of liver, spleen and glands, and febricula or isolated bouts of fever. The age distribution was: 1–5 years six cases, 6–10 years twenty-one, 11–20 years eleven, 21–30 years six, 31–40 years four. All were proved by xenodiagnostic methods with puppies. The Machado-Guerreiro reaction was tried in 17, and proved positive in 15, negative in two.

The five chronic patients were all between 29 and 60 years of age and all had cardiac arrhythmia.

The lower virulence of the disease in Chile is ascribed to the facts of immigration being less than in the countries bordering the Atlantic, and secondly of the disease being of longer standing in the western parts of America and the people being more resistant to the trypanosome [a lame explanation, if not mere tautology].

Three species of Triatomidae occur in Chile: *T. infestans*, *T. spinolai* and *Triatomaptera porteri*, but only the first seems to be really important as a vector. Forty per cent. of over 9,000 examined were found infected. As for reservoir hosts, dogs and cats are certainly such; 20·4 per cent. of 184 of the former and 4·4 per cent. of 136 of the latter were positive. Other animals, among them the llama, bat, fox, pig, and a small rodent, *Octodon degu*, have all been negative to examination.

The geographical distribution in Chile extends from the Province of Tarapacá to that of Curicó, and the majority of cases are found in Atacama and Santiago. It is quite common to find more than one member of a family infected. As regards age, the acute cases occurred equally in children and adults, the subacute more in children, and the chronic in adults. In studying the epidemiology, 1,445 Machado reactions were tried on inhabitants of the rural districts and 31·8 per cent. were positive. *H. Harold Scott.*

FALCÃO, J. de B. Caso agudo de tripanosomose americana observado em Santo Angelo das Missões, Rio Grande do Sul. [**An Acute Case of Chagas's Disease in Santo Angelo das Missões, Rio Grande do Sul.**] *Brasil-Medico.* 1943, Apr. 3–10, v. 57, Nos. 14–15, 179–82, 1 fig.

The author records the case of a girl of three years brought to him on account of a "swollen eye." There was painless palpebral oedema of the left eye, with erythema and mild conjunctivitis. The pre-auricular glands were enlarged on both sides. The case was quite straightforward and the diagnosis clear by the presence of *T. cruzi* in the blood-smear and was confirmed by xenodiagnostic methods. The course of the illness seems to have been satisfactory but nothing is said concerning the treatment adopted.

SIMÕES and TUPINAMBÁ had observed 24 cases in a few months (reported in 1942), 13 adults and 11 children, among 78 persons examined—a high proportion which was confirmed by xenodiagnosis. They also found two out of 13 dogs, one of 7 cats, and one of 11 armadillos positive. A few Triatomas, *T. infestans*, captured in the residence

of the patient recorded by the author showed four adults out of 10 positive, and two nymphs out of three ; one larva caught was negative.

H. Harold Scott.

VERSIANI, O. & JUNQUEIRA, M. Adenopatias chagasicas. Contribuição ao seu estudo. [**Adenopathy in Chagas's Disease.**] *Brasil-Medico.* 1943, Mar. 20–27, v. 57, Nos. 12–13, 147–52, 3 figs. English summary.

Examination of enlarged glands is the readiest means of diagnosis, if the direct blood examination is negative. The authors speak of "three different types of gland reaction." (1) Acute adenitis, very characteristic, with intense histiocytic proliferation, masking completely the normal gland structure, with polymorphonuclear infiltration constituting true micro-abscesses, more in the cortical region, and more or less extensive zones of necrosis and haemorrhage. Leishmania forms are often present, free or within macrophage cells, and flagellated forms in the medullary zones of the gland. (2) Primary regional adenitis, satellite glands round the first. (3) Secondary generalization of the adenitis. [These last two are described as different "types," but would seem to be of the same nature as the first and merely exhibiting different degrees of the same pathological changes. So-called Type II, for example, shows histiocytic proliferation, inflammatory infiltration, focal necrosis and haemorrhage, in fact, as the authors state, an attenuated form of Type I.]

There follows an account of a patient complaining of headache, giddiness, dysphagia and the signs of "mal de engasgo" [see this *Bulletin*, 1921, v. 18, 301 ; 1927, v. 24, 830] and enlarged glands, behind and above the right ear and others in the neck and groin. He was anaemic ; red corpuscles 2,815,000 per cmm., haemoglobin 35 per cent., colour index 0·6. *T. cruzi* was not seen in blood-smears, thick or thin. Ascaris and Ancylostoma ova present in the faeces. Four diagnoses were considered : Helminthiasis, Chagas's disease, Cyst of the ear, and Blastomycosis. Biopsy of the enlarged gland showed the changes detailed above and the diagnosis of Chagas's disease with helminthiasis was arrived at. Xenodiagnosis would have taken several weeks. Opportunity was taken to test the Intradermal Reaction of Montenegro in this condition, but it was negative, indicating that it is specific for leishmaniasis and is not given merely where there are leishmanial forms of *T. cruzi* present. *H. Harold Scott.*

DAVIS, D. J. **An Improved Antigen for Complement Fixation in American Trypanosomiasis.** *Pub. Health Rep.* Wash. 1943, May 14, v. 58, No. 20, 775–7.

The author describes the preparation of an antigen for complement fixation in Chagas's disease. This is made from cultures of *Trypanosoma cruzi* grown on a medium consisting of beef or horse meat infusion, 2 per cent. proteose peptone No. 3 (Difco), 0·7 per cent. sodium chloride, 0·5 per cent. dextrose, 2 per cent. agar, and 10 per cent. defibrinated rabbit blood. The *p*H is adjusted to 7·6 and sterile dextrose solution and blood added just before tubing. The medium is slanted in 25×200 mm. tubes (25 cc. in each), and after solidification of the slant it is covered with 25 cc. infusion broth containing proteose

peptone and dextrose in proportions given above. The tubes are inoculated with 0·5 cc. to 1 cc. of a culture of *T. cruzi* and kept at 25–28°C. for 10 days.

The broth containing the organisms is drawn off and centrifuged rapidly, after which the trypanosomes are washed three times with saline. Finally the material is washed and centrifuged in a 15 cc. graduated tube with saline containing merthiolate (1 : 10,000). The volume of packed material is measured, the supernatant fluid poured off and nine volumes of the saline-merthiolate mixture added. About 0·5 cc. packed trypanosomes are obtained from 20 culture tubes.

The suspension is frozen in dry ice and methyl cellosolve and allowed to thaw slowly, the process being repeated three times. The antigen is then ready for titration and is kept at 4–6°C.

It has been tested successfully with the sera of experimentally infected animals and of nine human cases of Chagas's disease in which complement fixation was positive in dilutions from 1 : 160 to 1 : 10. The antigen retained its stability for many months. *C. A. Hoare.*

MAZZOTTI, L. Estudios sobre *Triatoma hegneri*. I. Infección natural y experimental con *Trypanosoma cruzi*. II. Intentos de cruzamiento con *T. dimidiata*. [*Triatoma hegneri* : **I. Natural and Experimental Infection with** *T. cruzi*.] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1943, Mar., v. 4, No. 1, 53–6, 1 fig. English summary (4 lines).

"1. *Triatoma hegneri* Mazzotti, 1940, was found infected in nature with *Trypanosoma cruzi*. Experimental infection was also demonstrated.

"2. Experimental crossing between *T. hegneri* and *T. dimidiata* gave production to unfertile eggs."

IRIARTE, D. R. Infestación experimental por *S. cruzi* del Triatoma capitata-Usinger 1939. [**Experimental Infection of** *T. capitata* **with** *T. cruzi*.] *Bol. Laboratorio Clinica "Luis Razetti."* Caracas. 1943, May, v. 3, No. 10, 179–80, 1 fig.

CALDWELL, F. E. & GYÖRGY, P. **Effect of Biotin Deficiency on Duration of Infection with** *Trypanosoma lewisi* **in the Rat.** *Proc. Soc. Exp. Biol. & Med.* 1943, June, v. 53, No. 2, 116–19.

"*Summary.*—Biotin deficiency has been found to prolong the infection with *T. lewisi* in the rat. This effect can be negated by the administration of biotin to the deficient rat during the course of infection. Biotin appears to be instrumental directly or indirectly in the activation of the immune bodies in this infection."

LEISHMANIASIS.

POTENZA, L. & ANDEZE, P. J. Kala-Azar en el estado Bolivar, Venezuela. [**Kala Azar in the State of Bolivar, Venezuela.**] *Rev. Policlinica Caracas.* 1942, Nov.–Dec., v. 11, No. 67, 312–17.

Kala azar was first discovered in S. America by MIGONE, who described a case in an Italian who had resided in Paraguay for some

years [see this *Bulletin*, 1913, v. 1, 635]. Since then cases have been recorded from the Argentine, Brazil and Bolivia. In Venezuela, with which this paper is chiefly concerned, the first case was recorded by PONS and MARTÍNEZ in the state Guárico in 1941 [this *Bulletin*, 1942, v. 39, 607]. This led to a survey which included the examination of 842 specimens of liver removed by viscerotomy. In one of these, from a child ten years of age in the state of Bolivar, leishmania were discovered. Attempts to correlate these cases with the presence of Phlebotomus were not successful, for though these insects occurred in certain localities, in some of which there was evidence of the existence of cutaneous leishmaniasis, they could not be found in the districts in which the two cases of kala azar had been discovered. The authors admit, however, that further investigations are required before any conclusions can be drawn from these negative results.

C. M. Wenyon.

LAFUENTE, M. & GODARD, G. A propos de deux cas de kala-azar infantile. [**Two Cases of Infantile Kala Azar.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Mar., v. 20, No. 1, 33–8, 2 pls.

The paper describes two cases of kala azar in young children in the province of Constantine, Algeria. In both cases malaria had been regarded as the cause of the illness, but failure of response to anti-malarial treatment led to further examinations. In the one case splenic puncture revealed leishmania, while in the second case this parasite was discovered in large numbers in a film of the peripheral blood, along with the parasite of benign tertian malaria. The first patient responded rapidly to intravenous neostibosan, while the second was rapidly cured by this drug combined with quinine. It is pointed out that till neostibosan was administered to the second patient for treatment of the leishmania infection, quinine had failed to influence the malaria.

C. M. Wenyon.

FEVERS OF THE TYPHUS GROUP.

CIMMINO, V. Studio sull'andamento della febbre ricorrente e del dermotifo in Asmara nel quadriennio 1939–1942. [**On the Prevalence of Relapsing Fever and Typhus in Asmara in the Period 1939–1942.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1943, v. 2, No. 3, 85–93. English summary (5 lines).

The climate of the upper levels of Eritrea resembles that of many European cities, but the region is the site of diseases which militate against successful colonization, especially in the more thickly populated districts. The author has studied the question of the prevalence of two of the more important of these diseases, *viz.*, typhus and relapsing fever during the four years 1939–1942 inclusive. The diagnoses have been confirmed by the Weil-Felix reaction in the former and by finding the spirochaete in the latter. Analysing the returns month by month for Italians and for the natives during the four years he finds that there have been 141 European cases of typhus and 747 native cases, the greatest, 106 and 105 in February and March, 23 and 13 Europeans and 83 and 92 natives ; the lowest was 35 (9 European, 26 native) in

November. Of relapsing fever there have been 52 European and 651 native cases. In no month did the European total reach double figures during the whole four years. The greatest number of native cases was 109 in September, the next being 73 in August and 67 in October. Both diseases are to be regarded as endemic in Asmara and for both the vector is the louse, and the crowding together of natives in the wet and cooler months facilitates the spread of infection. The fact that more Europeans are attacked by typhus than by relapsing fever is peculiar and the explanation offered is not very satisfactory, that the non-medical man mistakes an attack of relapsing fever for a relapsing malaria and so does not attend for treatment. *H. Harold Scott.*

POTAPCHIK, J. A. **A Contribution to the Detection of the Spotted Fever Virus in the Reconvalescents.** *Arch. Sci. Biol.* Moscow. 1940, v. 58, No. 2. [In Russian 58–67, 3 charts. (11 refs.). English summary 67.]

In view of the existing controversy regarding the presence of the causative organism in convalescent cases of typhus fever, 54 patients who had recovered from the disease and showed a positive Weil-Felix reaction were tested during periods of convalescence varying from 1 to 18 months, by experimental infection of 89 guineapigs. Of these 57 were inoculated with the blood of the patients, 2 with the teased-up guts of lice fed on the patients, and 30 with brain emulsion from passage animals.

The results of examination of these animals were inconclusive, for in no case was it possible to reproduce a typical experimental infection with typhus fever. However, three strains could be interpreted as being positive but atypical since they showed only some of the characteristic symptoms, while the others were absent. Moreover, these strains were not readily passaged. It is concluded that convalescent cases do not play an important rôle in the epidemiology of typhus fever.

The author also notes that the Weil-Felix reaction may remain positive during convalescence for periods up to 39 months, with a titre of 1 : 200. *C. A. Hoare.*

BENHAMOU, E. Les rickettsia du typhus exanthématique et de la fièvre boutonneuse dans les cellules endothéliales de la moelle osseuse. [**The Rickettsiae of Exanthematic Typhus and Boutonneuse Fever in the Endothelial Cells of the Bone Marrow.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Dec., v. 20, No. 4, 309–13.

Smears of bone marrow obtained by sternal puncture from typhus patients show the following cytological changes :—(1) A great increase in the number of endothelial cells ; (2) A still greater increase in the plasmocytes ; (3) Numerous lymphocytes, 10 to 20 per cent. instead of the normal 3 to 5 per cent. ; (4) A fair number of megakaryocytes, 2 to 3 per cent. instead of 1 per cent. The percentages of the myelocytes, polynuclears, and erythroblasts, were correspondingly reduced.

The endothelial cells have been wrongly included as monocytes by previous workers.

Rickettsiae were found in the endothelial cells in the following forms :—(1) One or more morula-like masses which tend to be larger when only one is present in the cell. They are brighter-red than the cell nuclei and have a characteristic granular appearance ; (2) Punctiform granules like those originally described by RICKETTS ; (3) Large

homogeneous masses, one in each cell, corresponding to the "initial" bodies of trachoma; these have been found in only one case up to the time of writing.

These three forms correspond generally to the types recently described by GIROUD and PANTHIER in the lung cells of infected rabbits and suggest stages in the evolution of the Rickettsiae from isolated granular bodies to agglomerations of various types.

In boutonneuse fever the findings are of the same general type, but with minor differences, such as the presence of numerous eosinophile myelocytes, the tendency of some of the endothelial cells to undergo lysis, and the presence in these cells of small, rounded, rose-red granular masses.

The author claims that the search for Rickettsiae in sternal puncture fluid will enable an early diagnosis to be made in both diseases, and probably also in other Rickettsial diseases. *John W. D. Megaw.*

GALLARDO, F. P. Le bloquage du système réticulo-endothélial et l'infection par le virus du typhus exanthématique. [**Blocking of the Reticulo-Endothelial System, and Infection by the Virus of Typhus Exanthematicus.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Dec., v. 20, No. 4, 317–21.

The reticulo-endothelial system of several guineapigs was intensively blocked by intraperitoneal and subcutaneous injections of 1 per cent. solution of trypan blue. These animals reacted to intraperitoneal injections of *Rickettsia prowazeki* in exactly the same way as control guineapigs. *John W. D. Megaw.*

RENOUX, G. Réaction allergique chez l'homme dans le typhus exanthématique. [**Allergic Reaction in Exanthematic Typhus.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Dec., v. 20, No. 4, 314–16.

The occurrence of allergic reactions after the intradermal injection of typhus Rickettsiae into guineapigs recovered from typhus fever has already been recorded by CASTAÑEDA, and later GIROUD made a similar observation in human beings.

In the present investigation 0·1 cc. formolized rabbit-lung vaccine was injected into the dermis of one forearm, and formolized suspension of the lung of a healthy rabbit was injected into the other arm as a control. An immediate temporary reaction resulted in every case, probably due to the formol. In positive reactions the reading was made after 48 hours, and the appearance resembled that of the intradermal tuberculin reaction. The criterion of a positive reaction was the occurrence of a central palpable nodule and zone of necrosis surrounded by an erythematous zone, ranging from 1·5 to 6 cm. in diameter according to the intensity of the reaction.

Of 25 persons tested two to three weeks after defervescence, 23 were positive in degrees which corresponded generally with the severity of the attack. The attacks had been very mild in the two negative persons.

Of 41 sailors who were tested, seven gave positive results, and all of these had served in Africa for 7 to 10 years; they were also shown by Giroud's serum-protection test to be immune.

The results of the test were compared with those of the Giroud test in 94 persons who had received the last dose of a protective vaccine a month previously; they agreed in all but three cases, in one of which the intradermal reaction was positive and the Giroud test negative; in

the other two there was a very doubtful intradermal reaction and the Giroud test was positive. *John W. D. Megaw.*

GILDEMEISTER, E. & PETER, H. Fleckfieberstudien. III. Mitteilung: Ueber das Vorkommen und den Nachweis der *Rickettsia prowazeki* im bebrüteten und infizierten Hühnerei. [**The Site of Occurrence, and the Demonstration of,** *Rickettsia prowazeki* **in Infected Embryo Chicks.**] *Zent. f. Bakt.* I. Abt. Orig. 1943, Jan. 20, v. 149, No. 7/8, 425–8, 2 figs.

COX has described the Rickettsiae as being specially abundant in the yolk sac of the infected chick embryo and as being scanty in the chorio-allantoic membrane, embryo, and yolk. This finding is confirmed.

The structure of the yolk sac is described. In sections of the yolk sac the Rickettsiae are found in enormous numbers in the loose endodermal tissue; none are seen in the ectodermal tissue or in the endothelial cells of the blood vessels of the sac.

By the method of fluorescence microscopy described by PASS (1942) the Rickettsiae are specially well demonstrated. The method is not suitable for smear preparations because the yolk globules are highly fluorescent and so cause interference. Thin paraffin sections give the best results.

For yolk-sac smear preparations Castañeda's method of staining is recommended; it gives as good results as Machiavello's method and is more convenient.

The Rickettsiae are remarkably pleomorphic; small coccoid forms, rods, and threads, may occur in the same smear; sometimes one or other of these forms predominates. *John W. D. Megaw.*

VAN ROOYEN, C. E. & BEARCROFT, W. G. C. **Typhus Rickettsial Agglutination Tests in the Middle East Forces and Egypt.** *Edinburgh Med. J.* 1943, May, v. 50, No. 5, 257–72. [41 refs.]

In the important investigation described in this paper the authors have had the benefit of the help and advice of Col. J. S. K. BOYD, whose work on the serology of the fevers of the typhus group is well known.

In a large series of cases of epidemic louse-borne, and murine flea-borne, typhus the agglutination responses to three types of Proteus and two types of Rickettsia organisms have been determined.

The chief findings have been summarized by the reviewer from the data contained in the paper.

The places of origin and types of the disease were as follows:—

	Epidemic Typhus	Murine Typhus
Egyptian civilian patients from Cairo and Tanta hospitals (March to June 1943)	17	4
British and Allied troops stationed in—		
Iraq	6	1
Palestine and Syria	0	7
Cairo and Alexandria	6	3
Canal zone and other areas in the Middle East	4	23

From this distribution the authors were "sorely tempted" to speculate on the probability of the disease in cities being louse-borne and of the canal-area cases being disseminated by fleas. They also comment on the fact that all the cases from Palestine and Syria were murine.

Most of the tests were carried out in early convalescents but a few were made early in the second week of the illness. The titres were :—

Epidemic Cases.

	Proteus organisms			Rickettsiae	
	OX19	*OX2*	*OXK*	Epidemic	Murine
Range ...	$\frac{1}{300}$ to $\frac{1}{5000}$	Nil to $\frac{1}{3200}$	Nil to $\frac{1}{1600}$	$\frac{1}{200}$ to $\frac{1}{6400}$	Nil to $\frac{1}{1600}$
Average type of response	$\frac{1}{1600}$	$\frac{1}{400}$	$\frac{1}{200}$	$\frac{1}{1280}$	$\frac{1}{320}$

Murine Cases.

	Proteus organisms			Rickettsiae	
	OX19	*OX2*	*OXK*	Epidemic	Murine
Range ...	$\frac{1}{100}$ to $\frac{1}{16000}$	Nil to $\frac{1}{1500}$	Nil to $\frac{1}{3200}$	Nil to $\frac{1}{2000}$	$\frac{1}{50}$ to $\frac{1}{12800}$
Average type of response	$\frac{1}{1600}$	$\frac{1}{200}$	$\frac{1}{200}$	$\frac{1}{250}$	$\frac{1}{1800}$

In 10 cases, of which six were epidemic and four endemic, the *OX2* titre was 1–1,000 or over, and in five of these 10 cases the *OX2* titre was higher than that for any of the other organisms. One was "vaguely suggestive of tick typhus"; this was probably the case in which the *OX2* titre was 1–3,200, the *OX19* titre was 1–100, the epidemic-Rickettsia reaction was negative and the murine-Rickettsia titre was 1–50.

The corresponding reactions in control cases were :—

	Number of cases	Positive (1–100 to 1–200)			Positive (1–50 to 1–100)		No reaction to any organism
		OX19	*OX2*	*OXK*	Epidemic Rickettsia	Murine Rickettsia	
Healthy controls... ...	100	1	11	14	1	1	78
Malaria (B.T.)	20	1	1	5	1	2	11
Sandfly fever...	22	2	4	10	0	1	10
Other diseases (Typhoid, etc.)... ...	32	1	1	7	1	2	23

Low-titre reactions were common, especially in sandfly fever, and chiefly to *OX2* and *OXK*, whereas such reactions to Rickettsiae were exceptional and a titre of 1–100 was observed in only one case.

The Rickettsia reaction became positive about the same time as the Weil-Felix, but it reached a higher average titre and persisted longer. The Rickettsia suspensions, prepared by Prof. CRAIGIE of Toronto, were remarkably stable and consistent in their responses; the Proteus suspensions, though from a highly reliable source, did not always give consistent results. The one drawback to the Rickettsia reaction was that the readings were not easy to make.

The incidence of the disease and the case mortality were remarkably low among the troops. There was no case-to-case infection, and the louse-borne disease must have been acquired through contact with indigenous labourers.

[The authors appear to have assumed that when murine Rickettsiae were agglutinated in a higher titre than "epidemic" Rickettsiae the case was necessarily one of flea-borne typhus. The distribution of their cases supports this assumption, but some workers hold that louse-borne typhus may sometimes be caused by murine Rickettsiae. Two questions arise: (1) does the Rickettsia agglutination test differentiate between murine and historic types of Rickettsiae, and (2) if so, does the finding of a murine type of organism prove the case to be one of flea-borne typhus?

Recent work, including that of the authors, suggests that we can hope for a final reply in the affirmative to both questions. Extensive trials of the Rickettsia reactions are greatly needed for all the fevers of the typhus group to find out whether there is agreement between the agglutination responses and the types of the diseases as classified on clinical and epidemiological grounds.] · *John W. D. Megaw.*

REYNOLDS, F. H. K. & POLLARD, M. **The Employment of a Rickettsial Vaccine for Antigen in the Diagnostic Complement-Fixation Test.** *Amer. J. Trop. Med.* 1943, May, v. 23, No. 3, 321–3.

The authors state that it is recognised that the Weil-Felix reaction lacks specificity in the diagnosis of Rickettsial infections and that the complement-fixation test is rather specific in differentiating them.

They have found that commercial epidemic-typhus vaccine of the Cox type is a suitable antigen for use in the latter test. As little as 0·1 cc. of a one in five dilution of the vaccine in 0·85 per cent. saline gave satisfactory fixation with specific typhus antiserum.

The antigen gave negative results with antisera of other organisms, including Rocky Mountain fever Rickettsiae, *Proteus OX19*, *Bact. typhosum*, seven species of Salmonella, *Vibrio cholerae*, *Brucella abortus*, *Br. melitensis*, *Bact. tularense*, *Trypanosoma equiperdum*, and *T. cruzi*.

Only one positive result was obtained in 89 Wassermann-positive, and none in 32 Wassermann-negative, sera; the positive reaction was in a Mexican who gave a history of having had a typhus-like fever some years previously.

Sera from two cases of typhus were tested; one, taken on the 5th day, was negative; the other, taken on the 12th day, was positive.

A group of 32 persons were tested 12 days after the injection of the last dose of standard anti-typhus vaccine; all were negative. This finding was not regarded as showing absence of immunity, but it did show that false positives will not occur in vaccinated persons.

[This paper has been reprinted at pp. 433–5 of the July 1943 issue of the *American Journal of Tropical Medicine*, with the footnotes renumbered for greater clarity.] *John W. D. Megaw.*

STEUER, W. Ueber einfache Trocknung von Testbakterien für Agglutinationszwecke. [**Simple Methods of drying Bacterial Cultures for Agglutination Tests.**] *Ztschr. f. Immunitätsf. u. Exper. Therap.* 1943, Apr. 20, v. 103, No. 1, 26–34.

The author makes no claim for originality in the principles involved in the methods described; he points out that WILSON was the first to use bacterial cultures, dried *in vacuo*, for agglutination work.

The bacteria that can be dried without sacrifice of their agglutinability are *Proteus OX19* and the dysentery bacilli of the Shiga-Kruse and Flexner types. The organisms of the enteric group are not so suitable, because their H agglutinins are destroyed in the process of drying. Slope agar cultures of 24 hours' growth were used in all the methods.

One method was to wash off the culture with normal saline, centrifuge the suspension, mix the precipitate with equal parts of acetone and normal saline, then allow the mixture to dry, with occasional shaking, at room temperature or in the incubator. The resulting powder is sterile; it is mixed with normal saline and filtered through filter paper just before being used. Powders kept at room temperature for a year were found to have lost little or none of their agglutinability.

The simplest method of all was to pick up the culture with a loop which is also used to rub the growth on the surface of strips of firm, smooth, filter paper, 2 by 8 cm. and folded lengthwise. The rubbing is continued till the surface loses its moist shiny appearance and becomes matt, dry, and yellowish in colour. The treated papers are kept in sterile containers and can be used any time after a few days up to three months. A better method is to wash off the culture with normal saline, centrifuge the suspension two or three times at 3,000 revolutions every half hour, remove the supernatant fluid thoroughly and smear the precipitate on the filter paper, which is dried thoroughly and then stored in sterile corked test tubes or wrapped in envelopes made of a high grade of Cellophane. The envelopes can be sealed with a water-glass preparation. By adding 0·2 per cent. formol to the saline the organisms can be obtained in the killed condition.

To prepare a suspension for test purposes the strips are immersed in normal saline in a test tube; after occasional gentle shaking for five to ten minutes the bacteria float off.

With these papers agglutination tests can be carried out in the most primitive conditions. For example a drop of water can be added to a drop of fresh or dried blood on a glass slide; a small piece of the test paper is floated on the preparation and kept moving by the point of a needle; in positive cases the reaction is visible within a few minutes. The reaction is easier to observe if the strips are stained with alkaline methylene blue and thoroughly dried before being smeared with the bacteria. When stained strips are used the test can be made with a drop of fresh finger blood even without the addition of water.

The stained strips are specially suitable for the "dry-blood agglutination test" of Kudicke and Steuer, but they can be used with advantage in all the tests for which bacteria-coated papers are employed.

John W. D. Megaw.

GRUBMÜLLER, J. Neuritis nach Fleckfieber. [**Neuritis in Typhus Fever.**] *Ztschr. f. d. gesamte Neurol. u. Psychiatrie.* 1943, Mar. 10, **v. 175, No. 3, 403–8, 8 figs.**

Six cases of localized neuritis, in German soldiers suffering from typhus fever, are described. There were varying degrees of muscular wasting, paralysis, and sensory disturbances. The nerves specially affected were, respectively: (1) both ulnars; (2) left ulnar; (3) left upper brachial plexus and right ulnar; (4) both upper brachial plexuses; (5) left median; (6) right ulnar. In the sixth case there was also some tenderness on pressure over the sciatic nerves.

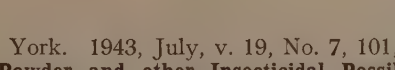
Similar forms of neuritis, probably also of toxic origin, occur in typhoid fever, but the author does not mention the possibility that pressure resulting from decubitus may be an important factor.

The onset was early in the course of the fever in three cases; in the others the condition was not detected till the temperature had fallen.

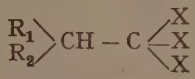
An interesting comment is that all the soldiers belonging to the same "group" as one of the patients were attacked by typhus.

John W. D. Megaw.

SOAP. New York. 1943, July, v. 19, No. 7, 101, 103 & 105. **Army Louse Powder and other Insecticidal Possibilities of Diphenyl Trichloroethane from which it is being manufactured.**

Dichlor diphenyl trichlorethane  is the active ingredient of the new U.S. Army louse powder. It is much more effective than the old formula which was not satisfactory under tropical conditions. [The old preparation contained pyrethrum, iso-butyl undecyleneamide and dinitroanisole.]

The new synthetic is highly effective for other insects besides lice: *e.g.*, it is stated that a mattress treated with it will remain free of bugs for 60 days. The material is covered by patents taken out in Britain by the Swiss firm Geigy A.G. B.P. 547,871 refers to compounds of the form:



where X is chlorine or bromine, R_1 is an organic radicle with at least 3 carbon atoms and R_2 is a radicle with at least 5 carbon atoms. The compound dichlor diphenyl trichlorethane can be made from chlorobenzene, chloral (or chloralhydrate) and sulphuric acid. In view of its formula it is surprising that this substance is non-irritating to man but very highly toxic to insects. It can be made up as an emulsion in water by a number of methods, of which examples are given in the patent specification.

B.P. 547,874 covers the same type of compound in powder form. This comprises dilution with an inert dust such as bentonite or bole, either by mixing or by application in a solvent and subsequent drying. Examples of solvents and methods are given. The final powders can be used directly as stomach or contact insecticides. One example, however, concerns deposition of the chemical on bentonite together with a wetting agent and sodium carbonate, then treatment with a

gelatin and potassium bichromate solution and finally drying. The powder obtained in this way can be wetted and diluted with water for spray application. *J. R. Busvine.*

WAR OFFICE. ARMY MED. DEPT. BULL. No. 25. 1943, July, 7. **An Experiment with the Lethane Body Belt.**

The "lethane belt" is understood to be a device which offers attractive harbourage to lice, and in which they tend to take up their quarters. It is doped with a non-volatile insecticide (lethane 384 special), which continues to kill them, even when the belt has been worn for long periods.

Large numbers of body lice were recovered from a typhus patient. Some were ground up and shown to be infective, by being injected into a guineapig. Others, after grinding, were placed between two layers of lethane belt. Even after 48 hours' intimate contact with the lethane, it was shown that the material was still infective.

[In the author's original report (of which this is a brief version) he remarks: "Every time we moved the garments louse faeces arose in the form of very fine red powdery dust. This material was highly infective and a few specks of it emulsified in saline and injected in a guineapig produced a sharp rise of temperature to 106°F. in 5 days." He states that one is "ill advised to sniff the faecal dust from such a garment. Indeed if any part of the cloth was rinsed in saline and the fluid injected in a guineapig immediate infection resulted, suggestive of massive amounts of virus."] *P. A. Buxton.*

PATIÑO-CAMARGO, L. Tifo murino en Bogota. Nota preliminar. **Murine Typhus in Bogotá.** [*Rev. Facul. de Med.* Bogotá. 1943, Mar., v. 11, No. 9, 503-14, 6 figs. on 2 pls. English summary.

Three types of Rickettsial fever have now been identified in Colombia. Two of these are regarded as belonging to the typhus group; they are louse-borne epidemic typhus and flea-borne murine typhus. The third is the tick-borne spotted or petechial fever which the author, following the usual North-American custom, places in a separate group.

In the present paper details are given of the isolation of murine Rickettsiae on three separate occasions in Bogotá.

(1) In October 1941: from the blood of a patient; from the brain of 6 out of 22 rats (*Rattus rattus* and *R. norvegicus*) caught in the place where the patient had been working; and from an emulsion of fleas (*Nosopsyllus fasciatus* and *Leptopsylla segnis*), caught on the rats.

(2) In April 1942: from the blood of a patient; and from fleas caught on rats from the warehouse where the patient slept.

(3) In July 1942: from fleas collected from rats at a residential school in which seven cases of fever had occurred between 29th April and 29th June, 1942. From at least 3 of 11 rats caught on the premises in July Rickettsiae were isolated. The rats were *R. norvegicus-decumanus*. The responses of different guineapigs to each lot of rat-brain, and flea, emulsion, varied from no apparent reaction to typical fever with orchitis. Six of the patients reacted to *Proteus OX19* at titres ranging from 1-100 to 1-800, and three to *Pr. OXL*: 1-100, 1-400, and 1-800, respectively. One patient was negative to *OX19* and four to *OXL*. *John W. D. Megaw.*

SINGH, B. **A Case of Typhus Fever.** *J. Indian Med. Ass.* 1943, May, v. 12, No. 8, 212, 1 chart.

An isolated case of fever, whose clinical features corresponded with those of Indian tick typhus, is described briefly. The place of occurrence was Meerut, about 40 miles from Delhi ; and the patient, a lady aged 40, was living in conditions in which " the possibility of infection through lice was inconceivable." There were dogs in the house, ticks were noticed in the rooms, and infection by ticks was regarded as probable. This was only the third case seen by the author during 30 years in Meerut.

The chief features of the attack were :—Duration of the fever, 14 days. Onset, on 25th September, 1942, was with pains in the back and joints. The temperature was 101·2°F. on the first day. It rose gradually to 104·2 on the fifth day and fell by gradual lysis from the ninth day. There was apathy on the fourth day, and low muttering delirium from the fifth day. The blood examination [day of fever not stated but probably about the sixth day] showed : total leucocytes, 12,800 ; polymorphonuclears, 82 per cent. ; monocytes, 1 per cent. ; lymphocytes, 17 per cent. The Widal and Weil-Felix reactions were negative. The rash appeared on the fifth day ; at first there were rose-red papules ; these became dark red, and faded after a week, leaving brown discolouration. The spots were profuse on the forearms and legs, fewer on the upper arms and thighs, and very few on the trunk and lower part of the neck ; there was none on the face. The rash resembled that of secondary syphilis. Clinically, this was a typical example of the tick typhus of India, and the epidemiological evidence strongly supports this diagnosis.

[The reviewer has seen a case of the disease in which infection occurred in the cold season in the same area. In this case, exceptionally, there was a local necrotic sore with associated lymphangitis at the site from which a tick had been removed just before the onset of the fever. In this case also a dog was probably responsible for transporting the tick to the patient.] *John W. D. Megaw.*

CAVAZZI, G. Segnalazione di cinque casi di febbre bottonosa sull' altopiano Etiopico. [**Record of Five Cases of Boutonneuse Fever in the Ethiopian Highlands.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1943, v. 2, No. 2, 5–9. [11 refs.] English summary (4 lines).

Boutonneuse fever has not hitherto been clearly identified in the Ethiopian Highlands. The five cases now described occurred in Gondar and Asmara [altitude about 7,500 feet].

The temperature rose gradually ; then there was continuous fever with slight morning remissions, and the termination was by rapid lysis. The fever lasted 12 to 15 days. There was a " tache noire " in the form of a black scab surrounded by a zone of erythema ; this appeared at, or even before, the onset, and persisted till the end of the fever or even a few days longer, leaving a red spot which soon disappeared and left no scar. Delirium was frequent at about the 10th day.

The rash appeared on the 2nd to the 5th day, first on the trunk, then all over the body, including the face, palms, and soles. In three cases the rash was papular, in two it was petechial and resembled the rash of louse-borne typhus.

In one case the Weil-Felix reaction was negative ; in the others the reaction did not become positive till the temperature had fallen. The titres to " X19 " were : 1–80 ; 1–160 ; 1–160 ; and 1–640.

The attacks were more severe than those described as typical of boutonneuse fever, though all the patients recovered.

[In the summary in English the disease is called tick-bite fever.]

John W. D. Megaw.

BAKER, G. E. **Rocky Mountain Spotted Fever.** *J. Amer. Med. Ass.* 1943, July 24, v. 122, No. 13, 841–50, 8 figs. [Refs. in footnotes.]

The author writes from the point of view of a physician with considerable personal experience of the disease in a part of Wyoming where severe and moderately severe cases predominate. He makes the interesting remark that " it is felt by some that in the future the name Rocky Mountain spotted fever will, of necessity, be changed." The name " tick fever " is frequently used in the paper as a synonym.

The description of the disease is on orthodox lines. Ticks are not usually found on the patients, but indurated sites of their previous attachment can sometimes be palpated ; the bite areas sometimes show discoloration and the local lymph nodes are at times palpable and tender. The petechial eruption first appears on the wrists and ankles within one or two days of the onset, but it may be difficult to detect till it spreads and becomes conspicuous. It is always more prominent on the extremities than elsewhere, and it may be absent, especially in mild attacks. Pigmentation persists at the sites of the spots and may be noticeable for several months whenever the skin is exposed to heat or cold. The temperature is sometimes distinctly remittent after the first few days, it falls by rapid or slow lysis. In very severe attacks it may remain normal till 24 hours before death. A secondary rise always suggests some complication. The pulse is relatively slow, the leucocyte count averages 12,000 to 15,000, but may be as high as 30,000.

In infected areas some protection may be afforded by tick-proof clothing, and by removing the clothes two or three times daily and making a thorough search for ticks, especially in the parts that escape friction by the clothing. Ticks should be removed immediately by gentle traction ; if this is not enough a small wedge of skin should be removed by scissors and the wound should be treated by phenol or iodine. Care should be taken to avoid crushing ticks, owing to the risk of infection through the unbroken skin. Ticks crawling on the body or even biting seldom attract attention. Scepticism is expressed with regard to the view that ticks cannot infect till they have been attached for several hours.

Treatment is on general lines ; fluids are given freely by mouth or, when necessary, by other routes. Convalescent serum appears to be useless, but hyperimmune serum of rabbits may be of value. The author believes that he has had success with intravenous injections of 0·3 gramme neoarsphenamine in 10 cc. of a 1–1,000 aqueous solution of metaphen, injected at the rate of 1 to 2 cc. a minute, and repeated every three or four days. The urine must be tested before each injection to ensure the absence of renal damage. About 30 cases have been treated in this way without a death, and with apparent benefit.

[On the other hand PARKER and other experts believe that intravenous medication of any kind usually hastens the death of experimental animals.]

The incidence and severity of the disease varies greatly from year to year ; for example the fatality rate in Wyoming was 12·3 in 1941, and 20·5 in 1942. The highest recorded was 31·7, in 1930, and the lowest 11·7, in 1933. *John W. D. Megaw.*

DAVIS, G. E. **American Q Fever : Experimental Transmission by the Argasid Ticks** *Ornithodoros moubata* **and** *O. hermsi.* *Pub. Health Rep.* Wash. 1943, June 25, v. 58, No. 26, 984–7.

Ornithodorus moubata, fed in the 1st nymph stage on a guineapig infected with *Rickettsia diaporica*, transmitted infection by feeding up to 428 days and harboured the Rickettsiae for 670 days. The tick did not transmit infection by feeding till the adult stage was reached. The progeny of infected ticks were capable of transmitting infection by feeding. This tick is widely distributed across Central Africa and in parts of South Africa, where it is the chief vector of tick-borne relapsing fever. It may be a natural vector of Q fever in these areas, though the disease has not been reported from Africa.

O. hermsi, infected as 1st or 2nd nymphs, transmitted infection up to 772 days and harboured Rickettsiae for 979 days ; in a smaller series of tests the progeny of this tick did not transmit infection by feeding.

Long periods of fasting did not diminish the virulence of the organisms in ticks. *John W. D. Megaw.*

MARIE, P. L. Maladies infectieuses des confins polonorusses : fièvre de Volhynie, fièvre d'Ukraine. [**Infectious Diseases in Poland and Russia : Wolhynian Fever (Trench Fever) and Ukrainian Fever.**] *Presse Méd.* 1943, July 17, v. 51, No. 27, 388–90. [11 refs.]

This paper contains a good summary of the literature dealing with the recent outbreaks of trench fever in the German army on the eastern front.

The articles dealt with by the author have already been reviewed in this *Bulletin* [1942, v. 39, 758 (JACOBI, J.) ; 1943, v. 40, 136 (KERGER, H.), 301 (ARNETH, J.), and 838 (v. BORMANN, F.)] but in view of the potential importance of the disease to military medical officers it is worth while to recapitulate the types of fever that have been encountered by German physicians. These are :—(1) The classical recurring type, in which paroxysms of fever occur every four to six days over periods ranging from ten days to two months or more. (2) The undulant type ; this is relatively uncommon. The periodicity of the spells of fever is the same as in the preceding type, but instead of sudden sharp paroxysms of fever there are recurring short waves lasting two to four days ; each spell has a gradual rise and a gradual fall of temperature. (3) The subfebrile type, with more or less continuous fever lasting one to three weeks, and occasionally followed by a further spell of fever of one to two weeks' duration, after a fever-free interval of two to three weeks. (4) The abortive type, in which there is slight fever for three or four days ; sometimes there is no fever at all. (5) The typhoid-like type, in which the curve resembles that of mild typhoid, but often this is preceded or followed by a period of recurring or undulant fever. (6) The typhus-like type, differing from the preceding in the sudden onset and the continued fever ; this is usually followed by a period of recurring or undulant fever. (7) The delayed-fever type, in which

there are pronounced symptoms for one to three weeks with occasional irregular rises of temperature which are hardly noticeable ; then one or other of the above types of fever follows.

The relative frequency of these types was very variable in the different outbreaks ; the recurring type was seldom predominant, more often the subfebrile or the larval type was the most common.

The pains showed similar variability ; severe shin-bone pains, most pronounced during the febrile periods, and greatly aggravated by warmth and decubitus, were usual, but by no means invariable. Often the pains were referred to other parts of the body and tended to shift from place to place. One constant feature was the absence of objective signs at the sites of the pains. No deaths have been recorded. In certain outbreaks a rash was seen in some cases ; this was macular and erythematous ; usually it was scanty and fleeting. No diagnostic test was available. *John W. D. Megaw.*

BARTONELLOSIS.

MERA, B. **Present Status of Human Bartonellosis.** *Bol. Oficina Sanitaria Panamericana.* 1943, Apr., v. 22, No. 4, 304–9.

[This is one of the best expositions of the subject within the knowledge of the reviewer ; it is at the same time succinct and thorough.] The epidemic in Colombia in 1936–39 which PATIÑO CAMARGO showed to be bartonellosis, after various suggested diagnoses of malaria, typhoid, typhus and brucellosis had proved erroneous, is referred to [see this *Bulletin*, 1940, v. 37, 271, 582]. Human bartonellosis is an infective disease, exclusively American and exclusively tropical, its distribution the Andes between latitudes 2°N. and 13°S., and the main vector Phlebotomus, *P. verrucarum* and *P. noguchii* in Peru ; seven species are found in Colombia but which is the chief vector is not known. Other arthropods may be capable transmitters, *e.g.*, *Dermacentor andersoni*, but are not proved natural vectors. *Suspected* reservoirs are many : Euphorbiaceae and several animals, domestic and other, *viz.*, horses, dogs, chickens, guineapigs, field mice ; the only certain one is man. The symptoms are divided into the two periods of haematic (systemic) and histioid (cutaneous), which may exist independently, or may coexist or may occur successively ; the pathogeny and pathology are described. The anaemia—the loss of red cells may be 200,000–300,000 per cmm. daily till the total count may be half a million or less—is ascribed to three factors (by HURTADO) : (1) The direct action of the organism growing in the corpuscles. (2) The tissue haemorrhages. (3) A haemolytic action of the Bartonella.

Control and prophylaxis are not easy. To deal with the reservoir is a problem at present unsolvable because we do not know what are the reservoirs, except man ; to destroy the larvae in cracks and crevices of walls and rocks is not feasible. There remains the breaking of the " Vector-man " link by attention to the following four rules :—

" (*a*) All individuals who work in the suspected zone should leave it before 6 p.m.

(*b*) All individuals who are compelled to spend the night in such a zone should carefully screen all the house, destroy the insects, and use repellents.

(*c*) The houses should be built in such a way that they get the most light possible, without dark corners and without any cracks or crevices. All the walls should be painted once monthly with creosote.

(*d*) Instruction of the public and improvement of the nutrition and living conditions are, of course, of prime importance in order to build up physiologic resistance to the infection." *H. Harold Scott.*

HOWE, C. **Carrión's Disease. Immunologic Studies.** *Arch. Intern. Med.* 1943, Aug., v. 72, No. 2, 147–67, 2 figs.

A painstaking study resulting in conclusions of considerable importance in this interesting disease. The primary purpose of the author was to examine the immunological response in relation to the course of the disease and the part played, if any, by agglutinins. The investigation was carried out in an endemic focus of the disease near Lima, Peru, and on patients in hospitals in Lima who had contracted the infection in endemic areas. Three species of Phlebotomus abound in verruga zones, *P. noguchii*, *P. peruensis* and *P. verrucarum*, the last being the chief vector. In all, 203 residents were examined, many of them new arrivals and most of them workers on an electric power project in the Santa Eulalia river valley. Blood cultures were made for each patient on the Geiman medium [this *Bulletin*, 1941, v. 38, 695], kept at room temperature (28±2°C.); blood examination included

Carrión's Disease.

Group	Blood Cultures	Agglutination	Eruption	Constitutional Symptoms	Past History of Carrión's Disease	Number in Group	Percentage of Series
1	+	+	...	+	—	16	7·9
2	+	+	+	...	—	9	4·4
3	—	+	+	...	—	6	3·0
4	+	—	+	...	—	15	7·4
5	+	—	...	+	—	3	1·4
6	+	5—, 1+	—	—	3+, 3—	6	3·0
7	—	+	—	—	3+, 3—	6	3·0
8	—	—	+	—	—	24	11·8
9	—	—	—	—	+	56	27·6
10	—	—	—	—	—	62	30·5
Number positive	49	38	54	19	62	203	100·0
Percentage of total ...	24·1	18·7	26·6	9·3	30·6		

total red and white cell counts, haematocrit and haemoglobin, and a film was stained for *Bartonella bacilliformis*, and serum was taken for agglutination tests with a formaldehyde antigen of four strains, the results being read after four hours at 37°C. followed by 12 hours in the refrigerator. The results are given in a table, reproduced above, the patients being divided by certain criteria into 10 groups. This table sets out clearly the findings and saves a long description.

The groups are as follows :—

(1) Patients with blood-culture positive for *B. bacilliformis* and agglutination positive, but presenting no eruption or past history of Carrion's disease, but with constitutional symptoms.

(2) Blood-culture and agglutination positive, eruption present, with or without constitutional symptoms, but not giving any history of the disease in the past.

(3) Blood-culture negative, agglutination positive, eruption present, otherwise as in group 2.

(4) Culture positive, agglutination negative, otherwise as in 2 and 3.

(5) Culture positive, agglutination negative, with constitutional symptoms, but no eruption or past history of an attack.

(6) Culture positive, agglutination variable, symptoms absent, and with or without history of previous attack.

(7) Culture negative, agglutination positive, no eruption or constitutional symptoms, with or without past history of the disease.

(8) Eruption present, but culture and agglutination negative, no constitutional symptoms or history of past attack.

(9) Past history of attack, but nothing else ; culture and agglutination negative, no constitutional symptoms.

(10) " Patients resident in endemic regions, with no past history or actual evidence of Carrión's disease, with blood culture negative for B. bacilliformis and negative agglutination test."

[This last is the largest group (62) and it is not very obvious, to the reviewer, at least, why they are designated " patients " at all, since they presented no signs, symptoms or past history of attack]. The author suggests that being residents in the endemic areas they had been more or less constantly exposed to infection and that they may have been naturally immune or have acquired immunity from a past inapparent infection. There is no evidence that agglutinins play any significant part in lasting acquired immunity ; group 9 is evidence to the contrary. The titre of agglutinins seems to reach a peak just before the eruption appears ; after its subsidence agglutinins are absent from the serum, though the organism may still be present in the blood and can be cultivated from it.

An experiment is being tried to immunize non-immunes (22 members of a military detachment stationed at infected localities in the Rimac river valley) by subcutaneous injection of formaldehyde vaccine, as used for the agglutination tests. One cc. was given and repeated in a week. In three weeks more all showed the presence of agglutinins (they had previously been negative) and after five months, though none had had any serious illness, *Bartonella bacilliformis* was cultured from the blood of 11 of them. This opens up the question of the possibility of at least modifying the severity of disease in those exposed to infection, though obviously vaccination does not prevent infection itself.

H. Harold Scott.

DELGADO BEDOYA, G. Verruga Peruana y malaria asociadas. [**Coincident Verruga peruviana and Malaria.**] *Rev. Facul. de Med.* Bogota. 1942, Mar., v. 10, No. 9, 593–602. [16 refs.]

In 1931 Professor ARCE reported malaria as a frequent complication of Carrión's disease and, second to this, bacterial infections, especially of the coli-typhoid group. HURTADO in 1938 remarked on the difficulties of diagnosis when the malaria masked the verruga, especially when, as is often the case, the verruga and malaria districts were the same. Splenomegaly, for example, was looked upon as a feature of Carrión's disease, whereas it was due to the malaria, enlargement of the spleen not being characteristic of the former disease. Blood changes are also complicated. Anisocytosis is more common in Carrión's disease, macrocytosis is not common in malaria, reticulocytosis is less seen in malarial anaemia, whereas leucopenia is more frequent. HURTADO, PONS and MERINO conclude that macrocytosis, hypochromia, reticulocytosis, marked anisocytosis and a normal relative leucocyte formula are points in favour of a diagnosis of Carrión's disease.

The author goes on to record the case of a man of 20 years in whose blood *P. vivax* was seen while he was suffering from an attack of Verruga peruviana. Incidentally the Wassermann reaction was negative in June, but when the Kahn test was tried a month later while the disease was still in progress a 3 plus was obtained. Later in his illness he suffered from a " dysenteriform colitis " with nine stools in the day [nothing is said of any bacteriological examination to determine the causative organism]. The patient was in hospital for four months.

H. Harold Scott.

YELLOW FEVER.

BRAZZAVILLE [AFRIQUE FRANÇAISE LIBRE]; RAPPORT SUR LE FONCTIONNEMENT TECHNIQUE DE L'INSTITUT PASTEUR EN 1941 [CECCALDI, J., Director]. 46–53. Fièvre jaune. [**Yellow Fever.**]

During 1941 a total of 780 vaccinations against yellow fever have been made at the Institute, using subcutaneous inoculations of a vaccine supplied by the Rockefeller Foundation made from tissue culture virus.

The " Dakar " strain of neurotropic virus has now reached its 414th passage in the brains of mice ; the Saleun-Ceccaldi strain from Brazzaville has reached its 98th passage.

Details are given of the examination of the sera from two Europeans suspected of yellow fever. One of these, from Donghila, gave protection against at least 1,840 mouse units. The serum of the second subject, vaccinated against yellow fever in Paris 31 months previously, showed no sign of any protective value. Tests were also made of four sera from natives collected in 1941. One of these, from Mayumba, protected against at least 1,280 mouse units.

During 1941 three Europeans and also three natives, four from Gabon and two from Cameroons, were diagnosed as cases of yellow fever as a result of a study of the pathological anatomy of the organs.

E. Hindle.

DUNNAHOO, G. L. **Insect Control on Aircraft.** *Soap.* New York. 1943, v. 19, No. 2, 111, 113. [Summary taken from *Rev. Applied Entom.* Ser. B. 1943, Aug., v. 31, Pt. 8, 166.]

The history of the control of mosquitos and other insects in aircraft is reviewed, and the setting up of disinfestation stations in Trinidad, Venezuela and Colombia and of inspection stations in Porto Rico, Haiti and Jamaica to cover all air routes from South America to the United States is reported. These stations were maintained from 1st November 1940 to 30th June 1941 by the United States Public Health Service and subsequently by Pan American Airways. The inspection reports for the first eight months are summarised. During this time, only two live mosquitos were found in the 208 aircraft from South America when they arrived at the port next after the one at which they had been treated. In experiments undertaken to improve the insecticide used in order to reduce the time of application and increase kill of more resistant species, it was found that the quantity of pyrethrum necessary to give complete kill of *Aëdes aegypti*, L., remained fairly constant regardless of the strength of insecticide prepared. To reduce the quantity of oil introduced into the aircraft, standard pyrethrum concentrate was used undiluted. Complete kill of mosquitos resulted when the pyrethrum extract was introduced at the rate of 5 cc. per 1,000 cu. ft. with power spraying apparatus producing a fine atomisation and the aircraft kept sealed for two minutes. When hand spraying apparatus was used, it was necessary to increase the rate to 8 cc. Pan-American Airways has ordered that all aircraft must be disinfested between all ports. The pyrethrum is projected with dichlorodifluoromethane, the mixture containing 2 per cent. pyrethrins, from a commercially available sprayer adapted as a refillable dispenser. With it, complete kill of mosquitos is obtained by operating for 3 seconds per 1,000 cu. ft. with an exposure of 2 minutes. The method of refilling is briefly described. [See also "Freon bomb," above p. 882.]

DENGUE.

SPADARO, O. Osservazioni sulla dengue. [**Observations on Dengue.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1942, v. 1, No. 5, 65–8. English summary (5 lines.)

In this paper the author gives his general impressions, based on personal observation of a large number of cases.

Dengue is endemic in Massawa, the capital of Eritrea. With the influx of susceptible newcomers the disease may assume epidemic dimensions; this happened to the Italian troops in 1935–1936. The disease is most common in the cool-weather months when Stegomyia mosquitoes are specially abundant. The symptoms are so varied that no uniform clinical picture can be painted. Immunity, lasting several years, is said to result from an attack; the histories of previous attacks that are given by some patients are discounted on the ground that they may have had some other disease. Patients who have afterwards lived up to two years in unaffected areas remained free from the disease even when exposed to great risks of infection.

Treatment is purely symptomatic; drugs that appear to have a favourable influence on the fever have no real effect other than that of delaying complete recovery.

[The author probably exaggerates the degree of immunity that follows an attack. Experiments on volunteers and many observations show that second attacks are relatively common though these are usually mild. Third attacks are infrequent, and some experienced workers believe that fourth attacks rarely or never occur.]

John W. D. Megaw.

PLAGUE.

MACCHIAVELLO, A. **Epidemiology of Plague in Ecuador.** *Amer. J. Pub. Health.* 1943, July, v. 33, No. 7, 807–11.

The author, a well-known writer on plague, does not support the very current theory that in the Andean region it is the guineapig (*Cavia aperea*) and its fleas, *Rhopalopsyllus cavicola* and *P. irritans*, which play the most important part in human plague. Undoubtedly the guineapig is one of the most susceptible of rodents to plague. It is a domestic animal in Uruguay, living in close household "contact with the Indian population which uses it as the principal source of meat." On the other hand the flea *R. cavicola* has not been recognized as a vector of plague from the guineapig to man, nor yet has *P. irritans* attained to any very important epidemiological rôle in the plague of other countries. One of the arguments which is said to favour the guineapig theory is that "in general domestic rats are scarce in the Sierra. . . . This statement has not been confirmed, but instead domestic species, especially *R. rattus* and *R. alexandrinus*, have been found to be universally distributed." The rat flea *X. cheopis* finds climatic factors in the zone 8,900 to 9,900 feet above sea level quite satisfactory, at least seasonally, for breeding. All that the guineapig may contribute to the plague epidemiology is "as a reinforcing factor in the infection" because it is a co-dweller with man, acts as a "flea-trap" and is extremely susceptible to plague. Thus the author contends, that "rural plague of the inter-Andean region conforms to the classic rat-flea mechanism, with changes imposed by the climatic characteristics under which the biologic activities of the rats and fleas are conducted. All the other phenomena foreign to this rat-flea mechanism should be considered accidental manifestations without any fundamental influence on it."

W. F. Harvey.

CHOLERA.

PANJA, G. & PAUL, B. M. **A Study of Invasiveness and Toxicity of Cholera, Paracholera and Saprophytic Vibrios in Animals.** *Indian Med. Gaz.* 1943, Apr., v. 78, No. 4, 190–91.

Cholera and cholera-like vibrios have to be differentiated as part of the technique of bacteriological diagnosis of true cholera. The further subdivision of the cholera-like vibrios is expressed in the qualifying

terms "paracholera" and "saprophytic." Animal tests have not bulked largely in the immediate differentiation of these vibrios, but the authors have applied them to that purpose. All three types of vibrio may be lethal to guineapigs by subcutaneous injection but, as is laid down in the summary, the important distinctions are :—

"1. *V. cholerae* and para-cholera vibrios, when injected subcutaneously in suitable doses into guineapigs, invade the general circulation, as a rule within 2 hours, but saprophytic vibrios found in Hooghly water do not do so so early. 2. No definite distinction can be made amongst the above types of vibrios by feeding in guineapigs, and by intravenous injection into rabbits. All the types of vibrios produce fatal results in the animals, the non-agglutinating vibrios producing a slightly higher percentage of fatality." *W. F. Harvey.*

GALLUT, J. Le complexe glucido-lipidique cholérique dans le vibrion et dans sa toxine. [**The Glucolipidic Complex in the Cholera Vibrio and its Toxin.**] *Ann. Inst. Pasteur.* 1943, Mar.–Apr., v. 69, No. 3–4. 123–6.

If it be admitted that endotoxin is of glucolipidic nature and exotoxin is protein then the author claims to have prepared a cholera toxin which contains both components. Endotoxin has been identified by BOIVIN with the glucolipidic complex of Gram-negative bacteria and as O-antigen. In his researches Gallut has extracted this glucolipidic substance from fresh type S vibrios of 18-hour growth on peptone agar. He has also obtained glucolipid from the toxin of a 4-hour growth on glucose medium, that is to say from the combined endo- and exotoxin. The yield of glucolipid is in this case always greater than that obtained directly from fresh vibrios. By this method of preparation a cholera toxin has been obtained in a minimal growth time and it contains both the glucolipidic and protein types. *W. F. Harvey.*

PANJA, G. & GHOSH, S. K. **Lethal Action of Potassium Permanganate on Vibrios.** *Indian Med. Gaz.* 1943, June, v. 78, No. 6, 288–91.

Potassium permanganate has long been used for the disinfection of well water, vegetables or fruit and stools during cholera epidemics. The present study gives precision to the activity of the permanganate by working out lethal dosage according to concentration, number of organisms present, and presence of organic matter. Well marked differences were found in the action of the permanganate on true cholera vibrios, non-agglutinating vibrios and unrelated organisms such as *Bact. typhosum.* The results are summarized : "Potassium permanganate in a high dilution of 1 in 10^6 in re-distilled pyrogen-free water is bactericidal to *Vibrio cholerae* and a still higher dilution of 1 in 10^9 is lethal to non-agglutinating vibrios. Much higher concentrations fail to kill even a small number of *Bact. typhosum.* In the presence of organic matter, a lower dilution such as 1 in 5,000 is necessary to kill the vibrios . . . . Fruits and vegetables artificially infected with cultures of *V. cholerae*, *Bact. typhosum* and *Bact. flexneri* can be effectively disinfected by soaking them in permanganate solutions of 1/5,000 to 1/10,000 dilutions for five minutes." *W. F. Harvey.*

[These findings are surprising in view of previous reports of similar work. GOHAR (see *Bulletin of Hygiene*, 1937, v. 12, 582) found that

1 in 1,000 permanganate was not enough to free artificially contaminated vegetables completely from *Bact. typhosum.* BERNARD (see *Bulletin of Hygiene*, 1938, v. 13, 400) concludes that it is impossible to sterilize salad leaves with permanganate without spoiling the appearance. VAN DEN BRANDEN and GEENS (this *Bulletin*, 1940, v. 37, 237) found that lettuce steeped in 5 per cent. permanganate for one hour was not sterilized. In view of these conflicting reports, and of the importance of the subject, it would seem desirable that further tests should be made.—*Ed.*]

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS.

SAVATEEV, N. I. **A Survey of Human Intestinal Protozoa in Sebastopol.** *Travaux Acad. Milit. Méd. Armée Rouge U.R.S.S.* Moscow. 1941, v. 25. [In Russian 394–8. English summary 398.]

In this survey, which is the first to be carried out in the Crimea, the author records the incidence (in 1937) of intestinal protozoa in Sebastopol, among two groups of hospital patients suffering from acute gastro-intestinal disorders, on the one hand, and in a group of school children, on the other hand. The total number examined was 401. While the latter group represents native Crimeans, the first two groups consist of newcomers to the country.

In dealing with the dysentery amoeba the author distinguishes between the race producing small cysts (under 10μ in diameter) to which he refers as *Entamoeba hartmanni*, and the race with large cysts (above 10μ) for which the name *E. histolytica* is reserved.

The results, estimated for the different groups (per cent.), were as follows: *E. histolytica* (*sensu lato*) 3·9–16·8 (comprising *E. histolytica* s.s. 6–16·8, *E. hartmanni* 3·9–12·9); *Endolimax nana* 10·2–22·0; *Iodamoeba bütschlii* 5·1–28·5; *Giardia intestinalis* 16·0–23·3; *Trichomonas hominis* 11·5–16·8; *Chilomastix mesnili* 7·6–15·4; *Balantidium coli* 0·4 (one case). The specific composition of the intestinal protozoal fauna in Sebastopol proved to be similar to that in other parts of the Soviet Union.

E. histolytica (*sensu lato*) was represented by both active forms and cysts in the hospital patients (first two groups) and by cysts alone in the school children. Since none of the patients showed clinical symptoms of amoebic dysentery and there was no evidence of haematophagy on the part of the parasites, the presence of active amoebae in the stools is attributed to the looseness of the faeces, while the persons themselves are regarded as symptomless carriers of *E. histolytica.* *C. A. Hoare.*

GNEZDILOV, V. G. **On the Commensal and Parasitic Forms of the Intestinal Protozoa in Connection with the Question on their Pathogenic Significance.** *Travaux Acad. Milit. Méd. Armée Rouge U.R.S.S.* Moscow. 1941, v. 25. [In Russian 370–92, 16 figs. (6 on 1 pl.) (36 refs.) English summary 392–3.]

It is well known that in spite of the cosmopolitan distribution and high incidence of infections with *Entamoeba histolytica*, cases of amoebic dysentery are comparatively rare and limited in their distribution. To

explain this discrepancy various hypotheses—which the author reviews—have been advanced. He adopts the views of REICHENOW [this *Bulletin*, 1932, v. 29, 121] and WESTPHAL [this *Bulletin*, 1938, v. 35, 586; 1939, v. 36, 288], according to which *E. histolytica* is normally a harmless commensal living in the lumen of the intestine but under certain conditions, found mainly in hot countries, the amoeba becomes a tissue parasite giving rise to the pathological conditions known as amoebiasis and amoebic dysentery. The factors favouring the passage of the amoebae from the lumen into the tissues have not been determined. It is suggested that the change in the habits of the amoeba may be influenced by the degree of structural and functional intactness of the gut wall, by the host's diet and state of nutrition, and by the symbiotic association of the amoebae with the intestinal bacteria. The author also believes that overheating, chills, fatigue, malnutrition, as well as acute intestinal disorders, traumata and intoxications, also tend to increase the virulence of the amoebae.

The normal form of *E. histolytica*, which is found in the lumen, is said to be the small one, generally described as the " precystic " form, while the larger form, which corresponds to the conventional active or " vegetative " amoeba, is regarded as the phase assumed by the lumen form when it invades the tissues. According to this interpretation, the large amoeba does not represent a necessary and indispensable stage in the life-history of this species. The two forms are said to differ not only in the morphology (size) of the active amoebae but also in their feeding habits, the small commensal (=lumen form) subsisting on bacteria, while the large pathogenic parasite (=tissue form) ingests erythrocytes. It is proposed to call the former phase *E. histolytica* forma *minuta* (mean diameter ca. 12μ), and the latter *E. histolytica* f. *magna* (mean diameter ca. 26μ).

The author maintains that some of the other human intestinal protozoa manifest morphological and biological differences of the same order. Thus *Entamoeba coli* is said to comprise two forms. One is the typical harmless commensal living in the lumen of the intestine and feeding on various micro-organisms and food-particles. The other form is identical with the amoeba formerly described as " *Councilmania lafleuri* "; it is larger than the normal amoeba, its feeding is osmotic (saprozoic), and, under certain conditions, it is capable of ingesting erythrocytes. There would thus appear to be a parallelism between this species and the dysentery amoeba, but whereas the " *magna* " form of *E. histolytica* is fully adapted to tissue parasitism, the corresponding form of *E. coli* manifests only a slight tendency to invade the tissues. The larger amoebae (mean diameter 28μ) are distinguished from the normal ones (18·5μ) by the name *E. coli* forma *magna*.

It is further noted that *Balantidium coli* may also consist of distinct strains. Some live in the lumen of the gut without producing any symptoms, while others invade the gut wall and give rise to balantidial dysentery. As in the amoebae, this transformation is associated with a change of diet: instead of bacteria, starch, etc., the ciliates ingest erythrocytes and other blood elements. The pathogenic form of Balantidium is said to be larger than the commensal phase, and is distinguished from the latter by the name *B. coli* forma *magna*. All the factors responsible for the development of tissue parasitism in this ciliate are not known but the author believes it may be due to a diminution of carbohydrates in the intestinal contents.

Finally, the author considers the case of *Trichomonas hominis*. It is pointed out that this flagellate may occur in the pus of liver abscess and is capable of invading the intestinal wall. Moreover, trichomonads found by the author in cases of bloody diarrhoea frequently contained ingested red blood corpuscles. In view of these facts this flagellate cannot be regarded as entirely harmless. The haematophagous forms, which were found to be larger than the typical ones, are designated as *T. hominis* forma *magna*.

In conclusion, and by way of summary, the author develops the general thesis that the intestinal protozoa considered above normally live as harmless commensals in the lumen of the gut, where they feed on its contents. Under certain conditions—especially prevalent in the tropics—they are capable of invading the intestinal wall, with the result that they become truly parasitic or even pathogenic. The tissue forms absorb nutrition by osmosis or they ingest red blood corpuscles ; and they can be distinguished from the lumen forms by their larger dimensions. While the pathological effects of *Entamoeba histolytica* and *Balantidium coli* are beyond doubt, the aetiological rôle of *Entamoeba coli* and *Trichomonas hominis* in various intestinal disorders, though suspected, stands in need of further investigations.

The paper is illustrated by figures showing the appearance of the protozoa in their commensal and parasitic phases. *C. A. Hoare.*

YONIS, Z. **Amebic Arthritis.** *Harefuah.* Jerusalem. 1943, Apr. 15, v. 24, No. 8. [In Hebrew 134–5. English summary 135.]

" A six year old child was suffering from inflammation of the elbow joint for six weeks.

" The joint was swollen, its movements were limited and painful. At times there was fever. The blood picture was normal. Mantoux, Kahn negative. B.S.R. increased. X-ray pictures of the affected joint were taken after the fourth and sixth weeks of the illness, without any pathological findings.

" Diagnostic features of the inflammation are discussed. . . . The puncture of the joint was avoided. In the stools trophozoites and cysts of Ent. histolytica were found. Under treatment with Emetine hydrochl. and Yatren all the symptoms disappeared and the child recovered."

HAUER, A. Erfahrungen mit einem neuen Mittel gegen Ruhr-Amöben-Infektion. [**Experiences with a New Drug in Amoebiasis.**] *Deut. Trop. Ztschr.* 1943, Apr. 1, v. 47, No. 7, 153–61.

The increasing incidence of amoebic infection and the uncertain action of yatren (quinoxyl) which is almost universally employed in Germany, together with many unsatisfactory features of several arsenicals in the treatment of amoebiasis, have rendered a quest for a new remedy advisable.

The present communication deals with the I.G. preparation 9659A—an arsenical-bismuth compound " Wia." It is claimed that the bismuth component prevents the uncontrolled absorption of arsenic from the damaged bowel and the sudden appearance of toxic manifestation. The " Wia " preparation is a white powder which is insoluble in water

and has a slightly bitter taste. The arsenical content is assessed at 15·7 per cent. : the bismuth at 37 per cent.

No neurological manifestations were introduced in animal experiments. The preparation is in tablets of 0·25 gm. each and the tolerated oral dose in rabbits was given as 2 gm. per kgm. Chemotherapeutic trials were carried out on amoeba-infected cats ; the dose was 30 mgm. per kgm. once or twice daily by mouth.

Since May 1942, amoebic dysentery cases, at first few, but later all new admissions were thoroughly treated with Wia. Directly the correct diagnosis had been established the patient was given a dose of calomel, then one tablet of Wia three times daily for 7–12 days. After November 1942, the dose was doubled. For two days nothing but fluids was given, after which the patient was permitted to move about and placed upon a light diet. Some were treated as ambulant cases after a pause of 3–7 days (in debilitated individuals 7–10 days) a second course on full dietary was instituted.

During and following the cure in 20 consecutive days the stools were examined microscopically.

As the result of these satisfactory trials Wia treatment has been meted out to troops in the field without any, except dietetic, restrictions.

Stools which were microscopically negative were cultured, but only in one instance with a positive result.

The patients were derived mostly from soldiers of the Afrika Corps as well as others from the Eastern front, who exhibited a lumen infection with *minuta* forms and cysts. A smaller proportion had more urgent symptoms with active stages of *E. histolytica* and ingested erythrocytes.

Seventy-eight patients with lumen infection and 10 with clinical amoebic dysentery were subjected to Wia treatment. In the early trials it was established that *minuta* forms and *E. histolytica* cysts rapidly disappeared from the stools. This was all the more significant since several were resistant cases which had relapsed after yatren-emetine courses. In a small number, however, *minuta* forms and cysts persisted for 8–10 days, and in one case up to 12 days, during the second Wia course.

Control experiments demonstrated no difference in response between those confined to bed and ambulant patients.

The preparation is easily tolerated and no disturbance of any kind was noted. On the other hand in diarrhoeic cases mild constipation was produced. It also has an action on other intestinal protozoa with the exception of Giardia. Thus after a few days' treatment, *E. coli* and cysts, *E. nana* and cysts, Iodamoeba and Trichomonas disappear.

Some failures were recorded, especially in cases of clinical amoebic dysentery with large vegetative forms of *E. histolytica* with erythrophagocytosis, but these responded to Wia combined with emetine.

There were also patients with *minuta* forms and cysts refractory to Wia ; these were afterwards cured by a yatren-emetine combination.

The local application of Wia to typical amoebic ulceration of the bowel by means of rectal retention enemata was unsuccessful. This is attributed to the relative insolubility of the product and its failure to penetrate amoebic lesions.

The author concludes that any apparent failures are due to specially virulent strains of E. *histolytica.*

The anti-diarrhoeic and sedative properties of the preparation on peristalsis are specially commented upon. *P. Manson-Bahr.*

OCHSNER, A. & DEBAKEY, M. **Amebic Hepatitis and Hepatic Abscess. An Analysis of 181 Cases with Review of the Literature.** *Surgery.* 1943, Mar. & Apr., v. 13, Nos. 3 & 4, 460–93; 612–49, 17 graphs & 24 figs. (1 coloured). [431 refs.]

A detailed and full, but not too full, account of the subject, giving much information, and well documented—in fact almost a monograph on a subject of perennial interest. The authors start with an abridged but adequate history of amoebic dysentery and its hepatic complications and sequelae, and then consider the incidence of amoebiasis in the United States, first in general and then from the aspects of age, sex and possible contributory factors. CRAIG estimated that "between six million and twelve million people in this country are infected with this parasite"—truly a wide range—but the present authors think even the upper limit far too conservative and that the figure is nearly twice this, 20–25 million, and that 4–5 per cent of these suffer from hepatic complications, *i.e.* approximately one million. Sex certainly seems to play a part, for of 1,789 cases of amoebic hepatitis and abscess collected from the literature, 1,703, or 95 per cent., were males, and of the authors' 181 cases, 157, or 86·7 per cent., were males. ROGERS and MEGAW found that 70 per cent. of some 400 cases analysed were in the third and fourth decades; of the authors' series the youngest patient was six years old, the oldest 70, and the average age was about 40 years. A few words are given as to the possible influence of alcoholism, trauma, diet, season, etc. They next discuss the pathogeny and pathology of the condition, noting that two main factors are intrahepatic portal thrombosis and infarction by the amoeba and its cytolytic activity. In the early phase there is a balance between regression to healing and scar-formation, and progression to abscess, and the balance is weighed down by the number and virulence of the organism overcoming the relative resistance of the host, or by the effect of bacteria, alcohol, trauma and the like.

The relative frequency of cardinal symptoms is detailed in the succeeding clinical section; in this analysis (181 cases) liver pain and tenderness were present in 86·7 per cent., fever in 84·5, enlargement of the liver in 72·9, loss of weight in 48, diarrhoea in 25·4, nausea and vomiting in 22·1, and jaundice in 10·5 per cent. The laboratory findings considered are the blood changes, the complement fixation test, examination of stools and X-ray appearances. As regards the first a table is given stating the numbers of leucocytes, of neutrophils, of erythrocytes and the percentage of haemoglobin, the highest, lowest and the average of each of these among the 181 cases. The complement fixation test is not very serviceable owing to the difficulty of obtaining a potent antigen. Sections follow on diagnosis and prognosis. Important factors in prognosis are the number of lesions in the liver, the existence of complications or of secondary infections, and the type of treatment used. SAMBUC in Haiphong in 1913 found that the mortality rose with the number of abscesses: 23 per cent. in those with single abscess, 45 per cent. in those with two, 90 per cent. with three, while of those who had four or more all died. In the authors' series, 124 had single abscesses and the fatality rate was only 10·5 per cent.; of 16 cases of multiple abscesses all ended fatally. The mode of operating also affects the prognosis; thus, of 24 cases with transpleural drainage eight were fatal, of 23 with transperitoneal drainage seven, while of 15 drained by the extraserous route only one

proved fatal [these figures are given as 33·3, 30·4 and 6·6 per cent. respectively].

The second part of the article deals with treatment. Aspiration is the procedure of choice if evacuation is necessary, but preliminary administration of emetine is important. In the authors' series among 80 who underwent open drainage there was a 22·1 per cent. mortality, whereas among 83 with " conservative therapy " there were only three deaths, 3·6 per cent. Open drainage is usually needed in those secondarily infected. The operation of open drainage is described and the complications which occurred were pleuropulmonary involvement 26 times in the authors' series, peritonitis 13, and pericarditis once only.

The article ends with descriptions of five illustrative cases : (1) Of satisfactory response to conservative therapy. (2) Abscess responding satisfactorily to emetine and aspiration. (3) An interesting case in a woman of 34 years, thought to be suffering from amoebic hepatitis, with characteristic clinical signs and X-ray showings. She responded at once to emetine, but a week later fever returned and a true abscess developed. (4) Amoebic abscess rupturing into a bronchus. (5) Abscess of liver opening into the right pleural cavity.

A valuable addendum to the article is the list of over 400 references.

H. Harold Scott.

CAMERON, J. A. M. **An Unusual Amoebic Liver Abscess.** [Memoranda.] *Brit. Med. J.* 1943, Sept. 11, 329.

A case of amoebic abscess of the left lobe of the liver in a Hindu woman is described. It was first treated with emetine hydrochloride injections, 10½ grains being given in 15 days. This was followed by a normal temperature but only for three days. The liver was then explored, first the right lobe without result and then the greatly enlarged left lobe, and 58 ounces of fluid, like thin pea-soup, were removed with a Potain aspirator. The temperature remained normal for seven days and the emetine was stopped ; the same signs and symptoms returned, however, and another 16 ounces of fluid were aspirated. As the patient continued to lose weight and strength open drainage was done and the cavity irrigated with eusol. The patient was discharged healed a month after the operation.

J. F. Corson.

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

GASPERINI, G. C. Considerazioni sulla epidemia di " Febbre Ricorrente " nei distretti di Asghedè, Tsellim Biet e Dembè Arcai (Tigrai Orientale) nell'ottobre-novembre 1940. [**An Outbreak of Relapsing Fever in the Tigrai District.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1942, v. 1, No. 3, 32–6.

The villages or townships mentioned are in the Tigrai district of Northern Abyssinia. Fever broke out there and a team was sent to investigate. The condition was found not to be " Mareb Fever," which is the local name for malaria. Blood-smears showed *Sp. recurrentis*. Ticks were not found but head- and clothes-lice were. Clinically, the

incubation period was about seven days, during which the patient might complain of slight headache. There was no rash, but a dry skin, with a subicteric tinge, enlarged and painful liver and spleen, and fever lasting 5–6 days ; after an apyrexial period of the same length, relapse occurred lasting 3–4 days. A single injection of Neosalvarsan (0·45 gm. for adults, 0·3 for adolescents, 0·15 for children) usually cleared up the symptoms. Two patients were recovering from typhus and their blood gave a positive result to the Weil-Felix test in dilutions of 1 : 5120 and 1 : 10,240 respectively.

In two villages the infection was severe and some deaths occurred. These were Addi Sardá and Addi Serannai. The number of cases there is not stated, figures being given only in percentages of the population attacked. In these villages these figures are given as 10·5 and 28·1 respectively, and the deaths as 7·5 and 15·6, from which it would appear that more than two-thirds of the patients in the former and half of those in the latter died. *H. Harold Scott.*

CHORINE, V. & CROUGUE, O. Virulence du sang du cobaye infecté avec le *Spirochaeta hispanica.* [**The Virulence of the Blood of Guineapigs infected with** *Spirochaeta hispanica.*] *Ann. Inst. Pasteur.* 1942, Nov.–Dec., v. 68, Nos. 11–12, 518–23.

The authors have made a comparative study of the virulence of the blood of guineapigs, infected with *S. hispanica* by the bites of *Ornithodorus erraticus*, at the beginning of the attack when spirochaetes are very numerous and during the intervals when the organisms cannot be found in the blood. The number of spirochaetes in the blood at the beginning of the attack is surprisingly high, and the inoculation of 1/10,000,000 cc. of the plasma of an infected animal is sufficient to produce infection. During the remissions when spirochaetes are not seen, the blood is much less virulent and it is necessary to use doses 100,000 to 1,000,000 times greater, in order to be sure of producing infection. The author then discusses the numbers of spirochaetes in the blood at the different stages of infection and estimates that during the remissions there are only 10 to 100 organisms in 1 cc. of blood compared with 1,000,000 to 10,000,000 during the attacks. Under these conditions the absence of spirochaetes during the "negative phase" may be explained as being only apparent, the result of the difficulty of finding organisms present in such scanty numbers. In addition the authors are of the opinion that it is very difficult to interpret the various atypical forms seen after silver impregnation, which have only a very remote resemblance to spirochaetes. *E. Hindle.*

CHORINE, V. Culture du spirochète de la poule. [**The Culture of the Fowl Spirochaete.**] *Ann. Inst. Pasteur.* 1942, Nov.–Dec., v. 68, Nos. 11–12, 524–7.

The author recommends a medium composed of unheated rabbit serum, diluted 1 in 5 with 0·5 per cent. peptone water. The latter is prepared by adding 5 gm. peptone to 1 litre of distilled water containing 7·5 gm. NaCl. After boiling for 5 minutes the *p*H is adjusted to 7·4–7·5, and the solution autoclaved at 120°C. for 10 minutes. The liquid is filtered whilst hot into flasks, each containing 150 cc. and these are autoclaved at 115°C. for 20 minutes. After mixing the serum and

peptone water in narrow test tubes, these are incubated at 37°C. for 24 hours to test for sterility, and then covered with a layer, 1 cm. thick, of sterile paraffin, and again tested for sterility.

When making the cultures a few drops of defibrinated blood may be added, or preferably laked blood, prepared by mixing 2 volumes of distilled water with 1 volume of defibrinated guineapig's blood. After standing for 2 hours, 10 per cent. NaCl solution is added in sufficient quantity to re-establish the ordinary molecular concentration of blood.

The mixture is then shaken, centrifuged at high speed for 20 minutes and the supernatant fluid filtered through a Chamberland L 2 or L 3 candle. When making cultures 1 part of this laked blood is added to 10 parts of the serum-peptone medium. The spirochaetes are then inoculated and the tubes incubated at 28° to 29°C.

Maximum growth is usually obtained about the 7th to the 10th day. Subcultures should be made about every seven days, although spirochaetes may be seen up to 21 days from the inoculation.

Cultures isolated 14 months previously, and having undergone 60 passages, were found to be fully virulent, and a fowl inoculated from this culture died of spirochaetosis five days later. *E. Hindle.*

SERGENT, A. & RICHARD, H. Cas unique de "résistance innée" à la spirochétose hispano-nord-africaine, observée chez un cobaye, sur plus de 3,000 inoculés. [**A Unique Case of Inborn Resistance in a Guineapig to North African Spirochaetosis.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Dec., v. 20, No. 4, 298.

DERRICK, E. H. *Leptospira pomona. Med. J. Australia.* 1942, Apr. 11, v. 1, No. 15, 431.

The author considers that the leptospira isolated in 1936 from the blood of a dairy farmer near Pomona in South Queensland [see this *Bulletin*, 1937, v. 34, 704] should now be given a specific name as it has been shown to be distinct from the three other known strains of Australian leptospira and also from eight foreign strains. Moreover, the clinical picture is fairly definite, resembling that of mud fever, seven-day fever, etc. Eighty cases of leptospirosis due to this strain have now been diagnosed in Queensland as a result of serum tests, and many others have been diagnosed clinically.

The name *Leptospira pomona* is proposed for this organism, which is distinguished from *L. icterohaemorrhagiae* and other leptospirae only by its serological reactions and its comparatively low virulence for man and laboratory animals. *E. Hindle.*

JOHNSON, D. W. **The Discovery of a Fifth Australian Type of Leptospirosis.** *Med. J. Australia.* 1942, Apr. 11, v. 1, No. 15, 431–3, 2 figs.

The description of a fifth type of leptospira in Australia, isolated from two cases of leptospirosis occurring in Queensland during 1940. One of these patients was a bullock driver and the other a meat inspector, and clinical details are given of the course of the disease. Both showed a sudden onset with headache and fever, gastro-intestinal upset and generalized muscular and joint pains. Leptospirae were

isolated from both patients and found to belong to a type serologically distinct from any of the known strains. The name *Leptospira mitis* is proposed for this organism. *E. Hindle.*

CLAPPER, M. & MYERS, G. B. **Clinical Manifestations of Weil's Disease with particular reference to Meningitis.** *Arch. Intern. Med.* 1943, July, v. 72, No. 1, 18–30, 4 charts. [15 refs.]

A description of 13 cases of Weil's disease occurring in the neighbourhood of Detroit, U.S.A., during 1940 and 1941. In two of these cases there was both clinical and laboratory evidence of meningitis; in seven there was an abnormal cellular reaction in the cerebrospinal fluid without clinical signs of meningeal irritation; and in one instance meningismus without pleocytosis of the cerebrospinal fluid. Lumbar puncture was not performed in two of the cases. All the patients presented clinical evidence of hepatitis during some phase of the illness.

The authors say that cell counts on the cerebrospinal fluid may reach 1,000 or more per cmm., with a predominance of polymorphonuclears early, and of lymphocytes later. The dextrose content is not altered. Yellow discolouration is common and is due, at least in part, to bilirubin. Marked retention of urea is common.

"Pericarditis, auricular fibrillation or disturbances of conduction may occur in hearts previously normal"; electrocardiograms are given of two of the cases. "The plasma prothrombin, although appreciably decreased in most instances, usually does not reach levels sufficiently low to account for the haemorrhagic manifestations. Anasarca, the result of hypoproteinaemia, may develop. Immuno-transfusions may be of value in treatment and are worthy of a more extended trial." *E. Hindle.*

YAWS.

GOLDMANN, C. H. & SMITH, S. J. **X-Ray Appearances of Bone in Yaws.** *Brit. J. Radiology.* 1943, Aug., v. 16, No. 188, 234–8, 12 figs.

This study is based on radiological examinations of 101 African civilians and soldiers in Sierra Leone (probably some of the latter also came from other West African Colonies). All lesions were in the tertiary stage. Active lesions were those in which acute osteitis, local pain and pyrexia were present. In inactive cases vague bone pains and malaise were predominant.

Positive Kahn tests, quick response to specific therapy and typical scars of secondary skin lesions were present in all cases.

The tibia was affected in 46, the fibula in 20 (both leg bones in 19), the femur in 13, the ulnar in 10, the radius in 7 (both forearm bones in six), and the humerus in nine. Among other bones the spine was involved in five cases, the skull in three and the pelvis in two.

The earliest active lesions in long bones were small oblong areas of translucency in the compacta, appearing within four weeks of the onset of symptoms. Often a worm-eaten appearance resulted. Periosteal changes were only present in association with changes in the compacta.

Without treatment lesions passed into the inactive stage. In this the bone was thickened and the medulla encroached upon, while the bony density was diminished in streaky lines so that demarcation between the compacta and medulla was lost. This picture might remain unchanged for years or slowly progress to that of " marble bone " in which the bone was massive but brittle. Slight trauma might result in fracture ; callus formation was then defective unless specific therapy was given. The majority of cases became quiescent without treatment. In some the compacta was thinned and the bone became bent. " Boomerang " deformity, and thin transverse bony bars were seen in the medulla. Specific therapy hastened the normal process to quiescence and the picture was then the same.

Trauma or intercurrent disease may cause active lesions to develop in bones in the inactive stage. In vertebral lesions there were eburnation, bony bridges and sometimes gummata. The disc was unaffected. In the frontal and other flat bones there were well defined areas of translucency in which healing started in the centre. Gummata were unusual in long bones with early lesions, except in children. Sequestra and periosteal changes were infrequent in flat bones.

During active bone growth, joints and epiphyses were often attacked, with considerable destruction, but symptoms were much less than would be expected.

While the authors say that yaws bone lesions could be imitated by syphilis they think that the absence of periosteal changes in yaws is of diagnostic significance. They say osteitis deformans is not uncommon among Africans and was often resembled by tertiary yaws. Stress is laid upon the high morbidity due to yaws in African soldiers.

[No mention is made of any cases in which " Boomerang " deformity actually developed under observation. A careful search among children who have not had yaws (*i.e.* who have no history, no scars and negative Kahn tests) would probably reveal some with such deformities.

The above differentiation of yaws and syphilis bone lesions can apply only to tertiary lesions. In secondary yaws bone lesions in children periosteal deposits are as frequent as, and even more extensive than, cortical lesions and may be the earliest radiological change. More destructive tertiary lesions than those described in this paper are frequently seen. Their absence may be due to the age grouping or limited communities from which the cases studied came.]

C. J. Hackett.

CUPI, N. Primi casi di pian riscontrati nel Governo dell'Amara. [**First Cases of Yaws observed in the Province of Amara, Ethiopia.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1943, v. 2, No. 3, 45–50. English summary (2 lines).

Yaws, says the author, is not a common disease in the Italian Colonies. In Amara, Harrar and Scioa not a case was seen in 1938 ; in Galla-Sidamo in that and the preceding years about 180 cases. In Somaliland, however, it is widespread, especially in the Bulo Burti Department, where 1,000 were reported in 1936, 1,502 in 1937, and " still more " in 1938. The author gives brief accounts of three cases seen by him in Gondar, all females, aged 17, 18 and 20 years. All gave a history of sexual intercourse, two of them shortly before the onset of symptoms, and, of course, all gave a positive Kahn reaction, but

the diagnosis was made because the primary lesion was extra-genital in each instance—left leg, abdomen and right hand respectively—the generalized rash resembled the primary lesion and the mucosae were not affected.

The rest of the paper is occupied with reiteration of the usual points of distinction between yaws and syphilis as stated in the text-books.

H. Harold Scott.

LEPROSY.

DANTAS, M. A lepra no Estado da Paraíba. (1°. Curso realizado entre 25 de Outubro de 1937 a 23 de Junho de 1938.) [**Leprosy in the State of Paraíba.**] *Rev. de Combate à Lepra.* 1942, Mar., v. 7, 5–17, 1 map.

The author divides his article into five sections dealing in succession with the origin and spread of leprosy in Brazil, reports of foreign authors, the history of the disease in Paraíba, measures to deal with it, and the results of a leper census in the State. Since all this is treated of in eleven pages the information is perforce sketchy.

Leprosy is believed to have been brought to Brazil by Portuguese colonists. By the end of the 17th century the municipality of Rio de Janeiro was becoming alarmed and asked for a special place to be set apart for dealing with them. Quotations from the writings of foreign authors are given; for example, according to Henri LELOIR, 1886, "Leprosy is especially common in the Provinces of Maranhão, Pará, Rio de Janeiro and Paraná, less in Minas and São Paulo. There are leprosaria in Baia, Côrte, Minas Geraes, Pernambuco and Rio de Janeiro." Z. PACHA in 1914, "Leprosy is still rife in Brazil, but is lessening." JEANSELME in 1934, "Lepers are found in two main localities, in the northern States of Amazonas, Pará, Ceará and Maranhão, and, in the south, Minas Geraes and São Paulo" and the total number was estimated at 70,000 to 100,000.

In Paraíba, the special subject of this study, in 1911 it was reported that lepers were very rare and one well-known doctor had not seen a single case. There was, however, a focus at Catolé da Rocha in the extreme north. For dealing with patients a leprosarium or better a colony establishment was proposed and a leprosarium of the colony type was erected at Rio de Meio, 8 kilometres from the town of João Pessôa, to house 130 patients, and in 1941 a Preventorium for 150 children of lepers. Between October 1937 and June 1938, as a result of four journeys to different districts in the interior, 50 lepers were found: 10 in the first survey occupying 46 days, 14 in the second (35 days), 12 in the third (28 days) 4 in the fourth; ten more were seen in other surveys between the trips to the interior. Of the total of fifty, 34 were males, 16 were females; 28 were white, 15 were half-castes and 7 black; none was under 10 years of age, 12 were in the second decade, 14 between 21 and 35 years, 13 between 36 and 50, 10 over 50, and one age unknown; 13 were urban and 37 lived in rural districts. It is thought that if a complete census were taken the total would be about two hundred.

H. Harold Scott.

MARTÍNEZ BÁEZ, M. Nota preliminar sobre la histopatologia de las manifestaciones cutaneas de la "forma de Lucio" de la lepra. [**Histopathology of the Skin in the Lucio Form of Leprosy.**] *Rev. Facul. de Med.* Bogota. 1942, Mar., v. 10, No. 9, 610–22, 8 figs. on 1 pl. English summary (5 lines).

Nearly a century ago, in 1851, a Mexican doctor, Rafael Lucio, described a special form of leprosy, a macular form, which has since been known as the "Lucio leprosy." Lucio himself said that the pathological histology needed description and the usual text-books omit details of the microscopic pathology of this form. In the present paper the author has rectified this omission, basing his remarks on biopsy specimens of five patients, taking tissue through the maculae. He finds in the dermis accumulations of "inflammatory cells" around the blood-vessels, dilatation of the vessels themselves, changes in the arteriole walls and, in some cases, foci of necrosis in the cell accumulations. The vessels appear as if wrapped round by these inflammatory cell masses. The capillaries are swollen by endothelial cells and perivascular proliferation. In appropriately stained sections the endothelial cells are seen to contain Hansen's bacilli, few or in globi. The perithelial aggregations consist of lymphoid elements, relatively large histiocytes, irregular in contour, but with homogeneous protoplasm. The sweat glands of the areas involved present narrowing of their ducts with almost complete obstruction by what looks like a mass of coagulated albuminoid substance and heaped-up small epithelial cells. In one of the preparations examined there was necrosis of the secretory part of the gland extending to the excretory duct and the reticular tissue of the dermis. Similar exudate may be seen around the nerve twigs in the deeper parts of the dermis. In the arterioles the lumen is narrowed and the inner surface is irregular from salients of endothial cell proliferation, and the wall may be thickened to many times that of the diameter of the lumen. *H. Harold Scott.*

LINHARES, H. Verificação da lepra murina na cidade do Rio de Janeiro. Sua distribuição geográfica e considerações endemiológicas. [**Murine Leprosy in Rio de Janeiro: Geographical Distribution.**] *Mem. Inst. Oswaldo Cruz.* 1942, Sept., v. 37, No. 3, 353–73. [59 refs.] English summary.

LINHARES, H. Contribuição ao estudo da patologia da lepra murina. [**The Pathology of Murine Leprosy.**] *Mem. Inst. Oswaldo Cruz.* 1942, Dec., v. 37, No. 4, 543–81, 24 figs. on 8 pls. [73 refs.] English summary.

HELMINTHIASIS.

MUKERJI, A. K. & GHOSH, B. K. **Deterioration of Ascaridol in Oil of Chenopodium.** *Indian Med. Gaz.* 1943, May, v. 78, No. 5, 234–5.

The standards of the British and U.S.A. Pharmacopoeias require that oil of chenopodium should contain not less than 65 per cent. w/w of ascaridol and that it should be kept away from the light in a cool place to prevent deterioration of its ascaridol content. Previous

workers are quoted who found that the ascaridol content of various samples of the oil varied from 45 to 70 per cent. The war has made it very difficult to obtain tetrachlorethylene and Mukerji decided to use oil of chenopodium instead, but first he tested a sample two years old by the standard B.P. assay method. In it he found only 49·16 per cent. of ascaridol. The authors then tested two other samples recently bought from two different makers. Each was divided into four portions which were put in brown or white bottles; one brown and one white bottle were kept in the light and dark respectively. A table shows the progressive deterioration of the ascaridol content in both samples, whether they were kept in the light or the dark, at a temperature ranging from 70 to 98°F. The deterioration was more rapid during the first year (1941) than during the subsequent two years.

G. Lapage.

MAPLESTONE, P. A. **Some Common Helminths of Man.** *Med. J. Australia.* 1943, July 10, v. 2, No. 2, 25–7.

BURT, J. C., LANE, C. M. & HAMILTON, J. L. **Report of a Case of Bilharziasis.** *J. Urology.* 1943, July, v. 50, No. 1, 68–70, 1 fig.

Remarking that bilharzia is infrequent in the United States, but that United States soldiers may be quartered in districts where it is prevalent and may there become infected, the authors record the case of a white missionary, a native of South Africa, who had been on leave for 15 months in the United States and developed painless haematuria. There were no other symptoms, but eggs of *Schistosoma haematobium* were found in the urine. Cystoscopy showed elevation of the bladder epithelium at three sites, one above the trigone and one on each lateral wall of the bladder. These were surrounded by nodules, one of which had ruptured, leaving a small irregular ulcer. The patient was treated with unspecified tartar emetic preparations. The source of the infection was not discovered.

The authors remark that the snail hosts do not occur in the United States, so that bilharzia cannot spread in that country unless some snail normally occurring in the United States proves capable of becoming an intermediate host; and this, they think, is very unlikely. [*Cf.* BLUM and LILGA, this *Bulletin*, 1943, v. 40, 552.] *G. Lapage.*

KRAKOWER, C., HOFFMAN, W. A. & AXTMAYER, J. H. **Portal-Systemic Collateral Veins in the Guinea Pig with Schistosomal Cirrhosis of the Liver and a Discussion of Congestive Splenomegaly.** *Arch. Pathology.* 1943, July, v. 36, No. 1, 39–50, 2 figs. [Refs. in footnotes.]

"The portal-systemic collateral veins which develop in the guineapig experimentally infected with Schistosoma mansoni are described. For anatomic and physiologic reasons, the view is expressed that in laboratory animals at any rate such anastomoses may be expected to function more efficiently in maintaining lower portal (splenic) venous pressures in the presence of factors leading to portal (splenic) venous hypertension than might be expected in man. To the extent that congestive splenomegaly is related to portal (splenic) venous hypertension, the failure to reproduce this condition experimentally is more readily understandable. The importance of these anastomoses in

schistosomiasis is stressed inasmuch as they serve as convenient pathways through which the parasites, their pigment and their ova can escape from the portal venous system."

FERGUSON, M. S. **Development of Eye Flukes of Fishes in the Lenses of Frogs, Turtles, Birds, and Mammals.**—*J. Parasitology.* 1943, Apr., v. 29, No. 2, 136–42. [16 refs.]

Diplostomum flexicaudum is a strigeid trematode found in the intestines of the herring gull in the Lake Michigan region ; its cercariae develop in species of Lymnaeid snails and, after leaving these, they penetrate fish, which are eaten by the gulls. The author found that the cercariae of *D. flexicaudum*, obtained from *Lymnaea stagnalis* and *L. palustris* were able to penetrate into, and grow into metacercariae in, the lenses of the eyes of tadpoles, frogs, turtles, chicks, ducklings, mice, rats, guineapigs and rabbits. Attempts to infect one pig and one sheep failed. The metacercariae thus obtained were normal in appearance and behaviour ; when those obtained from the lenses of a frog and a guineapig were injected directly into the duodenum of chicks they developed in one week in the intestines of the chicks into adult flukes, which produced eggs. In the lenses the cercariae cause opacity and sometimes liquefaction of the cortex of the lens ; blindness may result. In the eyes of fishes from which the lenses had been removed the cercariae did not survive.

There is probably plenty of opportunity for these cercariae to penetrate the eyes of human beings bathing in the lakes in Wisconsin, Michigan, Minnesota and the surrounding neighbourhood. But the author has found no record of this parasite in human beings, although non-strigeid flukes have been reported from the eyes of Europeans [WARD, 1918, *Amer. Encyc. and Dict. of Ophthalm.* (Wood), Chicago, v. 12, 9265, and SALZER, 1907, *Arch. Augenheilk*, v. 58, 19]. Possibly the eye secretions and the thickness of the eye tissues are effective barriers in man ; further, cercariae injected into mice and one guineapig did not survive nor migrate to the eye ; nor did large numbers of worms placed under the lower eyelid of mice and one rabbit penetrate the eye. The danger of infection of man is, therefore, rather remote, but cannot be excluded. If the cercariae did enter the lens their presence might not be detected for some time. The author suggests that, if the metacercariae died in the lens, little trace of them would remain and possibly repair of the lens and return to normal might occur.

G. Lapage.

SUMMERS, W. A. & WEINSTEIN, P. P. *Diphyllobothrium latum* **in Florida.** *Amer. J. Trop. Med.* 1943, May, v. 23, No. 3, 363–7, 5 figs. on 1 pl.

Three negro children in Jefferson Country, Florida, were found to be infected with *Diphyllobothrium latum.* A dog belonging to the family of two of these children was also infected. Two of the children were males, aged 4 and 14, and one was a female, aged 8. The sanitary conditions of the family which owned the dog, to which two of the children belonged, were bad and these children were in the habit of catching fish in local streams, cooking them over open fires, sometimes without cleaning them. None of the children or their families had ever been outside Florida State. Florida lies at the extreme south of

the North Temperate zone and *D. latum* is not usually regarded as being endemic in this zone. Literature is quoted which suggests that sporadic cases occurring outside the endemic areas are due to the importation of infected fish or to the entry of people who have become infected in endemic areas. The children were treated with oleoresin of aspidium and 22 scolices were recovered from one child, 11 from another; the results of treatment of the third child were not available. The worms recovered are described. It is suggested that the dog may act as a minor reservoir host.

[The recorded hosts of *D. latum* include man, the dog, fox, bear, mongoose, cat and various other Felidae, walrus, seal, sea-lion and pig.] *G. Lapage.*

MUKERJI, A. K. & MAPLESTONE, P. A. **The Treatment of Taeniasis.** *Indian Med. Gaz.* 1943, June, v. 78, No. 6, 282–3.

The authors have previously reported 75 per cent. of cures among 25 cases of taeniasis treated with carbon tetrachloride [this *Bulletin*, 1932, v. 29, 414]. They now record the results of treatment of more cases, making a total of 58, with this drug. One patient harboured *T. solium*. All the others were infected with *Taenia saginata* (one worm only except in one patient who passed 2 scolices).

A preliminary purge was not given. The patient had a meal of milk and bread at 7 p.m. the evening before treatment and no more food. Early next morning carbon tetrachloride in doses of 8–48 minims according to age was given [38 cases received the maximum dose of 48 minims (3 cc.)]. The only toxic signs noted were drowsiness and giddiness, which rapidly disappeared. Most of the patients had the carbon tetrachloride mixed with salts, but to nine patients the salts (magnesium or sodium sulphate) were given 1½–2 hours after the carbon tetrachloride. If the whole worm and its scolex were passed and no other strobila was seen the case was regarded as cured; some cases were followed for five months or longer. If a scolex was not passed, the cases were followed up and were pronounced cured if no segments were seen for three months or longer. The subsequent histories of 16 patients were not available. Of the remaining 42, 28 were cured by one treatment. Of the 14 uncured by one treatment seven were given a second treatment and six of these were cured by it; the seventh was not cured even by a third treatment (*i.e.* by three doses of 48 minims). The general result was a cure in 34 out of 42 cases, *i.e.* 80 per cent. The maximum dose (3 cc.) cured 24 out of 27 cases (89 per cent.).

Tetrachlorethylene cured only 54 per cent. of a total of 106 cases, including 26 previously reported by the authors [this *Bulletin*, 1938, v. 35, 282]. The maximum dose of this drug was 64 minims (4 cc.), other doses being given according to the age or undernourishment of the patients. Of the 106 treated, 45 did not subsequently report. Of the 61 remaining, 30 were cured by one treatment and 10 had a second treatment, three of these being cured by it. The maximum dose of 4 cc. cured 30 out of 46 cases. The only toxic sign was dizziness. Two subjects had experimentally induced infections with *T. solium*, one of them passed 4 scolices; all the others had *T. saginata* and all harboured single worms except one who passed four worms without scolices.

Hexylresorcinol was tried on 25 cases, including 10 previously reported by the authors [this *Bulletin*, 1933, v. 30, 205]. This drug was given in hard gelatine capsules early in the morning; no purge was taken on

the day of treatment, but if the bowels were not opened on that day salts were administered the following morning. The full dose of 1 gm. was given to 22 patients; the others received 0·75, 0·5 and 0·4 gm. The results were not traced in six cases. Of the remaining 19 the first dose cured five, and four more were cured by a second dose. Thus nine out of 19 cases were cured, a rate of 47 per cent.

Because carbon tetrachloride gave a cure rate of 80 per cent., while tetrachlorethylene cured only 54 per cent. and hexylresorcinol only 47 per cent., the authors conclude that carbon tetrachloride is the drug of choice for the treatment of taeniasis, provided that there are no contraindications. *G. Lapage.*

BARNETT, L. **Hydatid Cysts: their Location in the Various Organs and Tissues of the Body.** *Australian & New Zealand J. Surgery.* 1943, Apr., v. 12, No. 4, 240–48, 2 figs. [24 refs.]

PEREZ, B. Quiste hidatidico del pulmon. [**Hydatid Cyst of the Lung.**] *Bol. Asoc. Med. de Puerto Rico.* 1943, Apr., v. 35, No. 4, 129–42, 24 figs.

FINE, J. & LIVNY. **Filariasis in the Middle East.** *Brit. Med. J.* 1943, Sept. 11, 327–8.

The authors examined the blood of all the negro patients in a general hospital in Palestine, irrespective of the diagnosis attached to them, for the presence of microfilariae. Apparently wet films only were examined morning and evening; there was no opportunity to use stained films. Microfilariae were found in 12 out of 42 patients (28·5 per cent.); all these positives came from Uganda; since 30 of the 42 cases came from Uganda, the incidence among the Uganda soldiers examined was 40 per cent. In 10 out of the 12 positives only one larva was found. The authors think that the use of stained dehaemoglobinized thick films or the examination of the deposit from centrifuged haemolysed blood or the use of more blood might have increased the number of positives. Only two of the positives had clinical filariasis. The microfilariae in one case were diagnosed as those of *Loa loa*; in two other cases they were diagnosed as those of *Wuchereria bancrofti*. "It was clearly impossible to infer the species" in the others. Periods of from 10 months to two years and eight months had elapsed since the infected men had left Uganda and the authors conclude that their infections must have persisted as long as this. They agree that their investigation is limited, but think that the blood of natives recruited from filarial endemic areas in Africa should be examined and that infected recruits should not be sent out of the areas where they have been recruited, lest they infest the local arthropod vectors. *G. Lapage.*

GUY, W. H., COHEN, M. & JACOB, F. M. **Infection with Loa Loa.** *Arch. Dermat. & Syph.* 1943, June, v. 47, No. 6, 763–7, 4 figs.

The authors note two cases of *Loa loa* infection in American missionaries who had returned to the United States from the west coast of central Africa. The disease is very rare in the United States. One case only is reported. A woman aged 37 returned from the Cameroons in excellent general condition with a history of fugitive aching and

burning swellings on the ankles, arms, hands and wrists. A worm had been seen under the bulbar conjunctiva of the right eye. Four years later, after her return to the United States, a worm appeared under the bulbar conjunctiva of the left eye and the following year two appeared beneath the right upper palpebral conjunctiva. All these worms were removed. A microfilaria was also found in the blood about this time. One had been found in the blood seven years earlier in Africa. The patient had excellent health throughout. She was treated with mapharsen twice weekly and no Calabar swellings appeared during this treatment, but they reappeared when the drug was given only once a week. Antimony and potassium tartrate had no effect. *G. Lapage.*

ROSSI, G. Su di un caso di filariosi autoctona (filaria conjunctivae Addario) a localizzazione premammaria. [*Filaria conjunctivae* **in the Premammary Region.**] *Arch. Italiano di Chirurgia.* 1943, May, v. 64, No. 6, 456–64, 2 figs. [14 refs.]

F. conjunctivae Addario 1885 is rare in man. The author gives a list of fourteen cases (including his own) in seven of which the eye was the situation, in two the eyelids, in two the forearm, in one the peritoneum, in one a finger, and the present case in a woman of Vercelli, on the borders of Piedmont and Lombardy, 36 years of age, who had a small tumour situated in the subcutaneous tissue of the infero-external quadrant of the right breast. When excised this was found to contain a coiled-up immature female 8·1 cm. in length. [The author omits from his list BABUDIERI's case reported in 1937. (See this *Bulletin*, 1937, v. 34, 467).] *H. Harold Scott.*

RECTOR, L. E. **Factors influencing the Reported Incidence of Appendiceal Oxyuriasis.** *Amer. J. Trop. Med.* 1943, May, v. 23, No. 3, 369–72. [10 refs.]

Briefly reviewing the literature on this subject, the author points out marked variations in the records of the incidence of Enterobius in the appendix. He emphasizes his view that these variations may be due to the variable diligence with which surgical specimens have been examined. Some of the methods used by earlier workers are described. The author examined appendices from 160 females and 50 males above the age of puberty (arithmetical mean age 31·08) by gross examination on a slide, followed by examination of the mucosa with a lens and examination of some of the contents of the appendices. The necessity of examination of the contents is stressed.

Enterobius was found in 10 out of 210 appendices (4·76 per cent.). The ages of these 10 persons (8 females and 2 males) ranged from 15 to 33 (average age 22·7). The author thinks that diligent search of appendices would reveal an incidence of 15–20 per cent. in patients up to 15 years of age and of about 5 per cent. in patients above this age. *G. Lapage.*

SISK, W. N. **Effect of Phenothiazine on Intestinal Parasites.** *J. Amer. Med. Ass.* 1943, June 5, v. 122, No. 6, 357–60. [Refs. in footnotes.]

This paper deals mainly with the treatment of oxyuriasis with phenothiazine; the author finds that this drug is not effective for the removal of hookworms or Trichuris; and that comparatively large doses of it

(20–40 gm. given over five days) will remove some ascarids, but not all. The author finds that phenothiazine will remove ascarids from small children with less toxic symptoms than santonin. He was led to try the drug by reading the results of MANSON-BAHR's work (this *Bulletin*, 1941, v. 38, 516). [The author does not refer to similar tests with it made by MILLER and ALLEN (this *Bulletin*, 1942, v. 39, 781)]. Diagnosis and examination after treatment were made by means of the author's slight modification of the N.I.H. anal swab [*cf.* this *Bulletin*, 1942, v. 39, 780–81]. All the patients were white people.

The incidence of Enterobius infection was studied in three centres in North Carolina. In the first, 16 per cent. of 274 boys and girls aged 6–16 were positive by one swab only. Since 4–7 negative swabs are necessary to rule out infection, this figure of 16 per cent. must represent the absolute minimum infection rate. These children came largely from crowded homes and the lower economic levels. The second group examined were high school graduates aged 16–24 years; 115 were examined and only four were infected. The third group consisted of 62 children sleeping in wards close together at an orphanage; 38 per cent. were found to be infected after examination of two swabs. About two-thirds of the members of all families in which Enterobius was found were infected; in five of them all members of the families were infected. The author emphasizes that when Enterobius is found in a family, all the members must be treated.

Three schedules of doses of phenothiazine were devised. Schedule 1 was 8 gm. a day for five days (total 40 gm.) given to eight adults only. All the patients had nausea; four took the whole dose; four had to reduce it; four vomited several times and two became dizzy. Five children were given doses ranging from 4 gm. a day for a child of four years to 7 gm. a day for a child of 12 years. All had severe nausea and vomiting. All these cases were followed up for ten weeks, six or more Cellophane swabs being taken. All the swabs were negative. Five of the patients seen eight months later were still free from symptoms.

Schedule 2 was also a five-day course, as follows :—

TABLE 2.—SCHEDULE 2.

	Dosage, gm.	
	Per day	*Total*
Adults	4	20
Children		
8–12 years	3	15
4–8 years	2	10
2–4 years	1·5	7·5
Under 2 years	1	5

By this schedule 89 patients were treated and 68 showed no toxic symptoms. Eleven had nausea for two or more days; one was so nauseated that the full dose was not taken; one became dizzy; five vomited and in one there was a severe toxic reaction, which the author compares with the fatal case reported in the *Lancet* this *Bulletin*, 1942, v. 39, 193; see also JOHNSTONE, this *Bulletin*, 1942, v. 39, 560, for a case of anaemia following phenothiazine which was not fatal. In the author's case, a white girl aged 14, was given 4 gm. of phenothiazine daily for five days. Nausea and vomiting occurred from the fourth to the ninth days. On the sixth day she was pale and had blood in the urine, lasting until the 16th day. On the 9th day the mucosae were

pale ; there were no petechiae or jaundice. The erythrocytes numbered only 1,450,000, while the white blood cells were increased to 31,500. The haemoglobin was 30 per cent. and many nucleated red blood cells were present. After transfusion of 75 cc. of whole blood and treatment with iron, vitamins and liver, the erythrocytes increased to 3,500,000, the white blood cell count fell to 7,800 and the haemoglobin rose to 63 per cent. The patient recovered. Two of her sisters had similar but milder reactions.

Of the 89 patients in whose cases schedule 2 was followed, 74 per cent. were cured by the first course of treatment, 9 per cent. were not cured until after a second course, and 17 per cent. refused a second course.

Schedule 3 was as follows :—

TABLE 4.—SCHEDULE 3.

Adults and children over 6 years :
(*a*) 1 gm. a day for 6 days
(*b*) Rest 8 days
(*c*) 1 gm. a day for 6 days.
Children under 1 year : 0·25 gm. a day for the same period.
Children 1–6 years : 0·5 gm. a day for the same period.

No toxic symptoms followed in 62 children and four adults treated in this way. The effects were almost as good as those of schedule 2. The smallest total dose that caused toxic reactions was 20 gm. The author thinks that schedule 3 is an adequate and safe treatment for oxyuriasis and he recommends it for routine treatment. It is, he thinks, as effective as treatment with gentian violet and far better than any other treatment mentioned in the literature.

The chief symptoms of oxyuriasis are stated in their order of frequency as follows : nocturnal restlessness, poor appetite, nightmares, itching at the anus, nervousness, sleep-walking, indigestion, bed-wetting.

G. Lapage.

BERCOVITZ, Z., PAGE, R. C. & DE BEER, E. J. **Phenothiazine. Experimental and Clinical Study of Toxicity and Anthelmintic Value.** *J. Amer. Med. Ass.* 1943, Aug. 7, v. 122, No. 15, 1006–7.

The authors report on the effects of phenothiazine given orally to human patients and to rats. In 10 rats (with 10 controls) they attempted to produce toxic effects by giving them repeated doses in their food for 18 days until each rat had consumed 390 mgm. per kgm. rat weight. When the rats were killed no gross pathological lesions were found. Microscopical examination of organs showed no abnormalities except hypoplasia of all the cellular elements in the bone marrow of one rat only and normoblastic hyperplasia of the bone marrow in one other.

To 24 human patients 1½ gm. of phenothiazine were given three times daily for 10 days until 40 gm. had been taken. These patients had long-standing diseases (chronic ulcerative colitis, lymphogranuloma inguinale, or intestinal infestations with helminths or protozoa). None had acute infection, so that the authors conclude that any significant effects on the blood were due to the phenothiazine given. The blood and urine were examined before the drug was given, twice a week during the adminstration and once or more after it had been stopped.

Out of the 24 patients six showed no decrease in the number of erythrocytes, 11 showed a decrease up to 500,000 per cmm., four showed decreases of from 500,000 to 1,000,000, and three showed decreases of more than 1,000,000. Anisocytosis and poikilocytosis were frequent; erythroblasts were found in one blood smear only.

The haemoglobin level was studied in 23 patients. In five there was no decrease, in nine the decrease was less than 10 per cent. and in nine it was more than 10 per cent. The white blood cell counts were not significantly altered in any of the patients, with the exception of the appearance of an occasional myelocyte in two patients and of metamyelocytes in one. In one out of 23 patients there was a considerable amount of albumin in the urine, in six a trace, and in 13 none was present. Two patients had nausea and vomiting.

The effect of phenothiazine on intestinal parasites was studied on 44 patients. The doses for children of 2–6 years were $\frac{2}{3}$ gm. twice a day for 10 days (13 gm.), for children of 6–12 years the same dose three times a day for 10 days (20 gm.), and for adults $1\frac{1}{3}$ gm. three times a day for 10 days (40 gm.). For some patients these doses were repeated after a rest of two weeks.

Ten patients (children and adults) were treated for Enterobius infections, the criterion of cure being three negative NIH swabs [as many as seven may be required, *cf.* CRAM, this *Bulletin*, 1943, v. 40, 618]. Two patients were considered cured, the others were not. None of the other parasitic infestations was cured, although one out of three patients with *Entamoeba histolytica* had three negative stools after the treatment. The authors conclude that phenothiazine can produce toxic effects in an appreciable number of patients (*cf.* GRANT, below) and that it is not an effective anthelmintic or amoebicide. [No one familiar with the literature and use of phenothiazine would have expected it to have an appreciable effect on these infections with hookworm, *Ascaris lumbricoides*, *Taenia saginata*, *Schistosoma mansoni*, *Trichuris*, *Entamoeba histolytica*, *E. coli*, *Giardia*, *Iodamoeba*, *Dientamoeba* and *Endolimax*. The report prepared by the Agricultural Research Council entitled Co-ordinated Trials with Phenothiazine against Nematodes in Lambs (Imperial Agricultural Bureau, Penglais, Aberystwyth) clearly demonstrates the effective action of phenothiazine against certain nematodes.] *G. Lapage.*

MOST, H. **Studies on the Effectiveness of Phenothiazine in Human Nematode Infections.** *Amer. J. Trop. Med.* 1943, July, v. 23, No. 4, 459–64. [11 refs.]

The author records the successful elimination by phenothiazine of *Enterobius vermicularis* from 22 patients without any toxic effects and from 10 with mild toxic effects in only two individuals (haematuria in a girl aged 8 and mild haemolytic anaemia in a man aged 22), both of whom recovered in a day or two.

Phenothiazine of " commercial grade " was given in syrup, in gelatin capsules, spread as a sugary paste over crackers (biscuits) or in disintegrating tablets. Enterobius infestations were diagnosed and treatment was checked by NIH swabs. In one series of patients these swabs were taken daily for 10 days, beginning on the 14th day after the end of treatment, with subsequent swabs at about 10-day intervals until two months had elapsed after the cessation of treatment. In the

other series the criteria were the same except that the examination of swabs began one week after the cessation of treatment.

Reporting on the literature, the author notes that phenothiazine has been given to more than 200 patients without noteworthy toxicity. He tabulates 10 cases in which serious toxic effects (haemolytic anaemia or hepatitis) occurred and shows that, in all these cases, except one, the dose given considerably exceeded the dose of about 300 mgm. per kgm. of body weight, which he himself safely and successfully used for 22 of his patients. One of the two patients (a child aged 6 years) who showed the mild toxic effects noted above, received 1,100 mgm. per kgm. of body weight. The fatal case of acute haemolytic anaemia noted in the *Lancet* [this *Bulletin*, 1942, v. 39, 193] received, according to Most's calculation, 415 mgm. per kgm. of body weight.

The author suggests that the dosage of phenothiazine should be computed on the basis of the patient's body weight and that the total dose given should be the one which he used safely and successfully. Larger doses are not needed and may be toxic. The author's dose of about 300 mgm. per kgm. of body weight works out at the doses he gave to 22 of his patients, namely : a total dose of 5 gm. for children of 2–5 years ; a total of 7·5 gm. for children of 6–8 years ; a total of 10 gm. for children of 9–13 years ; and a total of 15 gm. for adults. These were successfully given over periods of 3 to 5 days in 22 cases ; they failed to cure in only three cases, two children and an adult, but these three were cured by second courses of treatment. The author thinks that the administration of the drug over a period of three days is apparently more effective than when it is given over a longer period.

The author, however, states his opinion that gentian violet is the drug of choice of Enterobius infestations and that phenothiazine should be given only to patients who cannot be cured by gentian violet or who are intolerant of it ; or in cases in which special clinical investigation is proceeding or which must, for any reason, be cured quickly. Phenothiazine should not be given to patients who cannot be observed once every three days. Very large doses of phenothiazine have been given without toxic effects, but idiosyncrasy occurs.

He also found that phenothiazine had no effect on infestations in the same and in other patients with *Ascaris lumbricoides*, *Trichuris trichiura*, *Necator americanus*, *Strongyloides stercoralis*, *Schistosoma mansoni*, *Entamoeba histolytica*, *E. coli* and *Giardia intestinalis*. [It is known that phenothiazine does not act on these species.] *G. Lapage.*

ELLIOTT, M. **Phenothiazine in the Treatment of Human Intestinal Helminthic Infestations.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1943, Sept., v. 37, No. 2, 163–4.

The author gave phenothiazine to over 70 West African natives and was impressed by the lack of unpleasant sequelae as compared with the results of treatment of 50 patients with santonin, oil of chenopodium or carbon tetrachloride. He gave total doses of 20–30 gm., spread over 4–5 days, without intolerance and says that very poor results are obtained if the whole dose is given in one day. The drug was given in 1 gm. tablets. Two of these were given three times a day after meals for 4–5 days. The dose must be strictly controlled, especially for children.

Results were assessed on examinations, begun at least a week after cessation of treatment, of " simple coverglass preparations " made

from the faeces; if two successive coverglass preparations showed no eggs the patient was considered to be cured. On this basis "cures" were obtained in the following infections: *Ancylostoma duodenale*, 26 out of 36; *Strongyloides stercoralis*, 5 out of 8; *Ascaris lumbricoides*, 9 out of 15; *Taenia saginata*, 4 out of 8; *Trichuris trichiura*, 5 out of 8; *Entamoeba histolytica*, 6 out of 9.

[Helminthologists would not consider the technique used to be adequate to check the effects of any drug on nematode infestations. See ELLIOTT, this *Bulletin*, 1943, v. 40, pp. 702–3, and the remarks there made.] *G. Lapage.*

GRANT, L. I. H. **A Case of Phenothiazine Poisoning in Sydney.** *Med. J. Australia.* 1943, July 10, v. 2, No. 2, 27–9, 1 fig.

The author emphasizes the danger of the use of phenothiazine for the treatment of Enterobius infections of man.

He describes the case of a girl aged 7½ years, admitted for headache, nausea and dizziness, with a history of Enterobius infection lasting four months at least, which had resisted treatment with santonin and calomel. Immediately after taking a course of treatment with phenothiazine, consisting of 2 gm. a day of this drug for seven days, given in powders three times a day, the patient lost appetite, became irritable and suffered from severe headache, tightness in the throat and chest and dizziness. The urine was a port wine colour. On admission to hospital two days after the administration of the last dose of the drug, the patient was very pale and slightly jaundiced, but there was no enlargement of the liver or spleen and no other abnormality. The erythrocytes numbered 1,810,000 per cmm. and showed anisocytosis, poikilocytosis and punctate basophilia; there were some nucleated red cells. The haemoglobin value was 40 per cent. and the colour index 1·1. The total number of white cells is not given, but the differential percentages were: neutrophils 62, lymphocytes 33, monocytes 1, myelocytes 3, and premyelocytes 1 (eosinophils are not mentioned).

The blood serum gave an indirect van den Bergh reaction. A transfusion of 400 cc. of fresh citrated blood was given by the constant drip method and next day there was general improvement. The erythrocytes now numbered 2,870,000, the haemoglobin was 57 per cent., the white blood cells 14,300 per cmm. Nucleated red cells amounted to 2 per 200 leucocytes and 8 per cent. of the red cells were reticulocytes.

No further special treatment was given; general measures included iron therapy and Dover's powder was given as a sedative. Three days later the erythrocytes numbered 2,750,000 per cmm., the haemoglobin value was 60 per cent., the white cells numbered 6,250 per cmm. and a differential count gave the following percentages: neutrophils 39, lymphocytes 54, monocytes 1, eosinophils 6. The improvement in the erythrocyte count and haemoglobin continued and eighteen days later there was complete restoration of the blood picture, the parents saying that the child had regained her usual well-being.

The author outlines gentian violet therapy, believing that phenothiazine is too dangerous, and that the former drug should be used. [He does not, however, mention the gastro-intestinal disturbances which gentian violet often causes.]

[For other papers on the efficacy and toxicity of phenothiazine see KUITUNEN-EKBAUM, 1942, v. 39, 192; DAVEY and INNES, *ibid.*, 861;

SCHNITZER, SIEBENMANN and BETT, *ibid.*, 862; HUMPHREYS, *ibid.*, 864; and ELLIOTT, *ibid.*, v. 40, 702.

MILLER and ALLEN, *ibid.*, 1942, v. 39, 781, compare the effects of phenothiazine and gentian violet on Enterobius, and CRAM, *ibid.*, 1943, v. 40, 618, records the view of American workers that gentian violet is the best drug for Enterobius, and gives its dosage. MOST (above) regards it as the drug of choice, and MACARTHUR (*Brit. Med. J.*, 1942, Jan. 31, 171; Feb. 28, 314) thinks it the most efficacious drug available for enterobiasis.

For a warning of the dangers of phenothiazine see the *Lancet*, 1943, Oct. 9, 453, and LAPAGE, G. (1943, *Brit. Encycl. Medical Practice. Interim Supplement*, pp. 13–16) where treatment with bismuth and by means of bismuth and barium meals is discussed. For treatment with bismuth see *Brit. Med. J.*, 1943, Sept. 25, 409; SIMPSON, G. (*ibid.*, 1929, Sept. 28, 604); and LOEPER (*Progrés Méd.*, 1920, v. 35, 339). For treatment by bismuth and barium meals see LAPAGE, C. P. [*Brit. Med. J.*, 1942, June 13, 738] and the Interim Supplement mentioned above.

Recent papers recorded in this *Bulletin* on the treatment of human infestations with phenothiazine refer to only a fraction of the considerable literature about this drug and its effects; see Bibliography of Phenothiazine as an Anthelmintic, [1942.] 6 pp., issued by the Imperial Bureau of Agricultural Parasitology (Helminthology), St. Albans; and Summary of Recent Abstracts on Helminthiasis, this *Bulletin*, 1943, v. 40, 571–8; 643–50.] *G. Lapage.*

RAPPAPORT, I. **A Comparison of Three Strains of** *Trichinella spiralis*. **I. Pathogenicity and Extent of Larval Development in the Musculature.** *Amer. J. Trop. Med.* 1943, May, v. 23, No. 3, 343–50, 3 charts. [13 refs.]

The author worked with three strains of *Trichinella spiralis* supplied to him by other workers. Two of these came from man, one from the pig. He emphasizes the necessity of accurate techniques when quantitative investigations in experimental trichiniasis are being made. The numbers of larvae given were counted on a ruled slide, the dilution-count method having been found unsatisfactory.

There appeared to be little or no difference among the three strains used in regard to lethal dose, extent of parasitism of the muscles and survival time of the mice experimentally infected (by stomach tube). Thus the variable results recorded in the literature, even when they are recorded by the same worker, are not likely to be due to variations in the strains of Trichinella used.

The lethal dose and survival time of animals infected have a limited value as criteria in work on trichiniasis. The author found it almost impossible to predict the survival time of the mice used, unless massive doses of larvae were given. The lethal dose of larvae for mice was variable, being, in a few cases, as low as 30 larvae per gm. mouse weight; in most mice it was 50–60 larvae per gm. mouse weight; but there was very little relationship between the size of the dose and the survival time of the mice, except with doses of above 100 larvae per gm. mouse weight. When less than 30 larvae per gm. mouse weight were given no mice died in the 60-day period following the dose; with doses of over 60 larvae per gm. all the mice died except two; with doses of 40 larvae per gm. many mice survived for the 60-day period. In the literature there is difference of opinion about the extent of muscle parasitism and especially about the relation of this to the dose

of larvae given. The author found that the proportion of larvae recovered (by artificial digestion of whole mice) to the number given varied considerably, but, when the larger doses of larvae were given, there was no trend towards an increase in the muscle parasitism, *i.e.* his work supports the view that the size of the dose of larvae has little or no influence on the extent of the muscle parasitism. *G. Lapage.*

RAPPAPORT, I. **A Comparison of Three Strains of *Trichinella spiralis*. II. Longevity and Sex Ratio of Adults in the Intestine and Rapidity of Larval Development in the Musculature.** *Amer. J. Trop. Med.* 1943, May, v. 23, No. 3, 351–62, 2 charts. [37 refs.]

Using the same three strains of Trichinella the author studied the length of life of adult Trichinellae in the intestine, their sex ratio and the rapidity of the larval development in the muscles. His results contradict the generally accepted view that the males are lost soon after the fertilization of the females, indicating that males persist after all females have been expelled.

Attention is drawn to the fact that the number of larvae given to the mice was counted directly on a slide and was therefore "almost exact." "Almost every worm" in the intestines of the mice was also counted by the following method: The mice were starved for 6–12 hours and the small intestine was removed, opened and put in 25 cc. of saline in a test tube; the caecum and rest of the large intestine were treated similarly in 10 cc. of saline. Refrigeration helped the separation of the worms from the mucosa and made the intestine more flaccid for examination, piece by piece, by compression between two slides and counting and determination of the sex of worms still attached to the mucosa. Most of the worms were in the fluid in the test tubes and these were counted and their sex determined individually by removal of 1 cc. portions of this fluid at a time.

The results indicated that there was little difference between the three strains of Trichinella as regards their longevity in the intestine, their sex ratio and the rapidity of development of their larvae. Larvae are laid rapidly by the females, most of them being laid between the 5th or 6th day after infestation and the 16th day, *i.e.* in 10–11 days. Counts of larvae digested out of the muscles indicated that most are probably laid in 4–8 days. The author reviews the literature and compares his work with that of others.

With small doses of larvae there was considerable variation in the proportion of larvae recovered to larvae given; when doses bordering on the lethal dose (for the lethal dose see RAPPAPORT, above) were given there was more consistency in this and also greater regularity in the longevity of the adults. After the first two weeks the adults were rapidly lost; after the first month they were usually absent from the small intestine, but they persisted much longer in the large intestine (up to the 54th day after infection). Sex ratios were always in favour of the females until the 16th–18th day (up to the 14th day they were of the order of 1 : 1·75 to 1 : 2·45). "Typically about 80 per cent. of adults remained in the small intestine until about the 16th day. Thereafter the females are apparently expelled rapidly, resulting in a sudden reversal of the sex ratio"; in many instances this resulted in complete loss of females, males only remaining. This contradicts the generally accepted view that males are lost soon after the females have been fertilized. *G. Lapage.*

LINNEWEH, F. & HARMSEN. Zur Allergie bei Trichinose. [**On Allergy in Trichiniasis.**] *Deut. med. Woch.* 1943, Apr. 30, v. 69, No. 17/18, 359–63. [34 refs.]

The authors consider that the essential symptoms of trichiniasis are allergic in nature; they discuss their reasons for this view in the first part of their paper. In the second part they describe the results of skin, complement fixation and precipitin tests done on 40 soldiers nine months after the onset of trichiniasis. These were done with five antigens prepared in slightly different ways. Three of them were made from the trichinosed flesh of rats (one preserved in 0·4 per cent. phenol, the others free from preservative) and the other two from the trichinosed flesh of pigs (both without phenol). The method of preparation is described. All but one of the antigens were dried and made up with normal saline before use, to dilutions of 1 : 500 and 1 : 10,000 for the skin reactions, and 1 : 2 and 3 : 10 for the complement fixation reactions.

The results indicated that the skin and complement fixation reactions are both useful for diagnosis, but that the precipitin reaction is not. For large numbers of patients the skin reaction is easiest and quickest; the maximal swelling and reddening appears usually in 15–20 minutes and has begun to disappear after 60 minutes. Some abnormal reactions among the patients are briefly described. Five of the slighter cases showed negative skin and complement fixation reactions when the tests were done, *i.e.* 9 months after the onset of the disease; but the moderately severe and severe cases were all positive, so that antibody had persisted in these for 9 months. The authors think that variable results obtained by earlier workers are doubtless due to variability in the constitution of the antigen and that their own work also shows this.

The antigens derived from rat muscle were better than those obtained from pig muscle. The authors cannot support GAASE's view [*Bulletin of Hygiene*, 1943, v. 18, 210] that pig antigen is better, nor his theory that the best results follow the use of antigen from the same species or from a similar species; but they point out that one of their pig antigens, which was sent to them by Gaase, probably was no longer fully active when they used it. *G. Lapage.*

DICKEY, E. S. **Trichinosis: as viewed by Different Interests.** *J. Amer. Vet. Med. Ass.* 1943, May, v. 102, No. 794, 359–64.

This paper is reviewed in *Bulletin of Hygiene*, 1943, v. 18, p. 724.

STERN, K. & DANCEY, T. E. **Acute Psychosis associated with Trichiniasis.** *Canadian Med. Ass. J.* 1943, Mar., v. 48, No. 3, 235–7.

This paper is reviewed in *Bulletin of Hygiene*, 1943, v. 18, p. 724.

DEFICIENCY DISEASES.

AYKROYD, W. R. **Vitamin B_1 Deficiency in Infants.** *J. Ass. Med. Women in India.* 1943, May, v. 31, No. 2, 41–6.

"(1) Infantile beri-beri or vitamin B_1 deficiency usually occurs in breast-fed infants aged 1–6 months. The disease may be of an acute

fulminating type or more chronic in nature. In less acute cases the signs are not easy to distinguish from those caused by other forms of malnutrition in infants and by common infantile sickness.

" (2) The clinical condition in the infant may be caused by lack of vitamin B_1 in the mother's milk or by a toxic factor in the milk produced by deranged carbohydrate metabolism.

" (3) The therapeutic test provides the surest method of diagnosis. Two milligrammes of pure vitamin B_1 may be given subcutaneously.

" (4) The mother's diet is usually one composed almost entirely of machine-milled raw rice. She may sometimes, but not always, herself show signs of beri-beri.

" (5) It is suggested that doctors and others concerned with the care of infants in India should be on the watch for the condition and observe the effect of giving vitamin B_1 to infants, particularly breast-fed infants, which are not thriving and show clinical signs for which there is no other obvious cause."

DAS GUPTA, B. K. **Parenchymatous Keratitis following Riboflavin (Vitamin B_2) Deficiency.** *Indian Med. Gaz.* 1943, Apr., v. 78, No. 4, 198–200.

After some introductory remarks about riboflavin and induced riboflavin deficiency, etc. [which are not altogether accurate] a single case is cited of a 30-year-old Hindu complaining of photophobia and rapid failure of vision of the left eye. On examination the right eye was normal in all respects. The cornea of the left eye was " almost totally opaque " but no vascularization was noted. Slight angular stomatitis and slight fissuring of the tongue is said to have been present ; scrotal skin normal, W.R. not done. Riboflavin 4 mgm. was given daily. The angular stomatitis and tongue condition cleared quickly, the cornea after a month. The diagnosis here was parenchymatous keratitis [whether associated with riboflavin deficiency and whether influenced by exhibition of riboflavin is another matter].

H. S. Stannus.

NAPIER, L. E. & CHAUDHURI, R. N. **Recurring Pellagra Syndrome in a Myxoedematous Subject.** *Indian Med. Gaz.* 1943, Apr., v. 78, No. 4, 183–6, 4 figs. on 1 pl.

The case now reported " is of special interest in that there is no evidence either of dietetic deficiency or of any bowel condition that might be associated with malabsorption " [of nicotinic acid]. " The condition was not cured by giving a well-balanced diet but required niacin in maximal doses ; the striking thing was, however, that cure could be equally effected by the administration of thyroid extract up to 10 grains daily." Previous attacks of pellagra had been treated with thyroid and nicotinic acid ; the last relapse with typical symptoms of pellagra responded to thyroid. The authors conclude " There appears to be strong evidence that cure of the symptoms of pellagra can be effected either by niacin or by thyroid." *H. S. Stannus.*

SPRUE.

INGELFINGER, F. J. & MOSS, R. E. **The Motility of the Small Intestine in Sprue.** *J. Clin. Investigation.* 1943, May, v. 22, No. 3, 345-52, 4 figs. [16 refs.]

An abnormal small-intestinal pattern has been demonstrated by X-ray studies in both tropical and nontropical varieties of sprue, but it has been shown that these changes may occur in other chronic deficiency states. In two patients, one with tropical the other with non-tropical sprue, the small-intestinal motor activity was studied by means of the balloon-kymograph method.

The first had extreme emaciation, distension and cramps so that three ileostomy operations had been performed in an effort to relieve these symptoms. When records obtained from the sprue jejunum were compared with the normal, striking differences were noted. Whereas the normal upper small intestine prevents the standard balloon from filling with more than 10–30 cc. of air under pressures averaging 20 cm. of water, the bowel in sprue offers less resistance to increases in the volume of the balloon. The lack of intestinal resistance to distension is roughly proportional to the severity of the disease.

In the first case cited pressure of 5–9 cm. of water sufficed to inflate the balloon with 75 cc. of air, but in the second 45 cc. of air was maintained by pressure of 10–15 cm. of water.

The record of the normal upper small intestine is characterized by large (L) and smaller (S) waves which may be superimposed or occur independently.

When first studied, no spontaneous L waves were observed in either case, whilst the S waves produced smaller excursions than normal.

Treatment over a short period with individual fractions of the vitamin B complex produced little change in the tracings, but prolonged administration of the whole complex did cause some improvement in the records; these were hardly commensurate with the clinical results. On the other hand acetyl-beta-methylcholine chloride stimulated intestinal motility, prostigmine being without effect.

In the first patient posterior-pituitary solution, adrenal-cortical extract and desoxycorticosterone produced no changes.

These observations suggest that in sprue the nervous apparatus of the small intestine fails to liberate active acetylcholine. It should be added that balloon records of intestinal motor activity are significant only if correctly used to interpret changes in the intestinal lumen.

P. Manson-Bahr.

ABICHT, I. & KUHLMANN, F. Ueber das Verhalten der Wasserresorption bei der einheimischen Sprue. [**Water Absorption in Sprue.**] *Klin. Woch.* 1943, May 15, v. 22, No. 20–21, 353–6, 7 figs.

This paper mainly concerns itself with water-absorption in sprue. One well-marked case in a woman of 50 provides the text, and it is quite clear from the description, as well as from the bone changes in the illustration, that the case is not one of tropical sprue, but of idiopathic steatorrhoea.

The arguments are involved and by no means easy to follow. The main evidence rests on the interpretation of X-ray examination of the gastro-intestinal tract. The presence of abnormal quantities of fluid

in the bowel is explained by stagnation, the result of diminished absorption. It is not due, as has been suggested, to active, inflammatory secretion into the lumen of the bowel. This is the explanation of the exaggerated water content of the faeces, as well as of the dehydration of the patient. *P. Manson-Bahr.*

HAEMATOLOGY.

TROWELL, H. C. **Dimorphic Anaemia. Deficiency of Iron associated with Nutritional Macrocytic Anaemia.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1943, July, v. 37, No. 1, 19–40, 3 figs. [22 refs.]

The classification of tropical anaemias on a simple basis of associated diseases resulted in certain difficulties especially concerning treatment, some cases of nutritional macrocytic anaemia improving with iron and some of hookworm anaemia benefiting from liver. An attempt was therefore made to define the deficiencies present, to ascertain what factors in the diet and what concomitant disease might give rise to these deficiencies, or might destroy blood or retard its regeneration. Of 134 cases of anaemia, classified by mean corpuscular volume and mean corpuscular haemoglobin concentration, 27 were found to be macrocytic orthochromic (nutritional macrocytic anaemia), 63 macrocytic hypochromic, 27 normocytic hypochromic, 5 microcytic hypochromic (pure iron deficiency), 11 normocytic orthochromic and 1 microcytic orthochromic. The cases in the second and third groups suggested a mixture of nutritional macrocytic and iron deficiency anaemias. Some were severe, requiring treatment both by iron and liver, which, given serially, induced a double reticulocyte response; in others one deficiency predominated and the cases recovered, albeit slowly, when the major deficiency was corrected. For these dual deficiency anaemias, the name "dimorphic anaemia" is suggested since in such cases the peripheral blood shows two types of cell, the marrow a mixed type of erythropoiesis, and two dietectic factors are concerned in the aetiology and treatment.

[This paper is the first of the author's series on the same subject but owing to delays in communications was not the first published. See this *Bulletin*, 1943, v. 40, 329, 484.] *F. Murgatroyd.*

VENOMS AND ANTIVENENES.

PENNA SOBRINHO, O. Anaveneno e o seu valor na produção de soros anti-ofidicos. [**Anavenin for the Production of Antivenenes.**] *Brasil-Medico.* 1943, June 19 & 26, v. 57, Nos. 25 & 26, 272–3. English summary (5 lines).

In 1936 the Ezequiel Dias Biological Institute started preparing snake antivenenes, using a stock venene consisting of venom 10 gm., glycerin 80 cc., physiological saline 120 cc. The venom was a mixture of *Bothrops jararaca*, 6 gm., *B. alternata*, *B. neuwiedii*, *B. atrox* and *B. jararacussú*, each 1 gm. For diluting Crotalus stock

venom NaCl 1·5 per cent. was used. For preparing the antivenene horses were injected subcutaneously twice a week with gradually increasing doses, starting with 0·075 mgm., and increasing till at the twenty-fifth inoculation 350 mgm. was the dose. The Bothrops antivenene was quite satisfactory, that against Crotalus was not, so the injections were continued, thirteen more being given and the final dose was 1,000 mgm.

The decision was then made to test the value of anavenin for immunizing horses. For making this a mixture of titurated venom, 13·3 gm., glycerin 333·3 cc., formol 13·3 cc., saline to 1,000 cc. was kept at 37°C. for 40 days, then filtered. A horse was inoculated with this subcutaneously, starting with 0·005 mgm., and at intervals of 3–4 days at first, later a week, till at the forty-second inoculation after eight months 0·45 mgm. was used. This was slightly modified later. Thus, for anti-Crotalus serum anavenin of 10 gm. of venom, 330 cc. glycerin and 10 cc. of formol was used and the period of immunization shortened by eight weeks. An antiserum was obtained of which 1 cc. neutralized 1,000 m.l.d. for a pigeon. For anti-Bothrops serum this method is not necessary and shows no advantage as the ordinary one is quite satisfactory, except that during the immunizing, abscesses are common complications; they are usually sterile and are just as frequent when anavenin is used. *H. Harold Scott.*

MARTINS, A. V. Anaveneno escorpionico. [**Scorpion Anavenene.**] *Brasil-Medico.* 1943, June 5–12, v. 57, Nos. 23–24, 248–51. English summary.

"It is possible to reduce in a great extent the toxicity of scorpion venom solutions by adding 1 per cent. formol and keeping in the ice-box at 4°C. for approximately 40 days, without affecting its antigenic value. The anavenom so prepared can be used with great advantages in the preparation of the antiscorpionic serum."

BEASLEY, B. T. **Arachnidism. Case Report of Arachnidism with Death and Autopsy Finding.** *Southern Surgeon.* 1942, Oct., v. 11, No. 10, 737–41. [39 refs.]

A boy, aged 16 years, was admitted to hospital at midnight and died next day at 8.50 a.m. He complained of vomiting and thirst, pains in the legs and intense abdominal pain. On admission he was pale, with moderate cyanosis of the lips and finger tips, restless and semi-comatose. The abdomen was rigid but not tender. A blood-count showed 35,100 leucocytes per cmm. of which 81 per cent. were neutrophiles. His temperature was 100°F. A specimen of urine was not obtained. He was given an injection of morphia (gr. ¼) and atropine (gr. 1/150) and 1,000 cc. of normal saline, containing 2·5 per cent. glucose, subcutaneously. His temperature rose to 106·5°F. and at 8 a.m. he had a convulsion lasting 2 or 3 minutes.

Post-mortem examination showed no naked-eye pathological appearances. Sections of the kidney showed swelling of the epithelial cells of the tubules and extravasation of erythrocytes. The cerebrospinal fluid, taken at autopsy by cisternal puncture, contained 210 cells per cmm., mostly lymphocytes.

From the symptoms and the history that he collected insects and had several live specimens of the "Black Widow" spider (*Latrodectus mactans*), a diagnosis of arachnidism was made. [See also this *Bulletin*, 1936, v. 33, 401; 1942, v. 39, 720.] *J. F. Corson.*

DERMATOLOGY AND FUNGOUS DISEASES.

ENGLISH, P. B. & GREY, L. P. **Sap Dermatitis and Conjunctivitis caused by the Wild Fig** (*Ficus tumila*). *Med. J. Australia.* 1943, June 26, v. 1, No. 26, 578–9.

Several men employed in clearing an area in which *Ficus tumila*, the wild fig, was growing presented themselves suffering from an acute dermatitis with blistering, affecting chiefly the forehead, the cubital fossae and genitalia, together with conjunctivitis. Common symptoms were those of coryza, irritation of the nose and throat with nasal discharge, and photophobia with lachrymation. The skin lesions ranged from blotchy erythema to vesicles as large as a pea. The arms probably infected the face and eyes when wiping away the sweat, and the genitalia involved probably by the sweat running down the body. Desquamation would follow in a week or so. The eye lesions might be so severe as to include denudation of the corneal epithelium with resultant nebulae. Experimental work proved that the sap of the *Ficus tumula* was the cause. *H. Harold Scott.*

FRANKS, A. G. & TAYLOR, H. G. **Cutaneous Blastomycosis complicated by Meningitis.** *Arch. Dermat. & Syph.* 1943, July, v. 48, No. 1, 88–90.

"A case of North American blastomycosis complicated by meningitis, caused by B. dermatitidis, is reported. The clinical, histologic and postmortem observations are presented. The mycologic studies are enumerated."

CATANEI, A. Sur des changements de caractères culturaux de *Nocardia madurae*. Etude morphologique et expérimentale. [**On Variation in the Cultural Characters of** *Nocardia madurae*. **Morphological and Experimental Study.**] *Arch. Inst. Pasteur d'Algérie.* 1942, Dec., v. 20, No. 4, 299–304, 2 pls.

From old strains of *Nocardia madurae* (*Actinomyces madurae* (Vincent 1894)), propagated for many years on Sabouraud's glucose-agar medium, the author isolated a variant strain which could be propagated in subculture apparently without reversion to the characters of the parent culture. As compared with the parent strain, the variant grew more rapidly and more luxuriantly, was more deeply pigmented, less adherent to solid media, showed greater friability and fragmentation of the mycelium, and developed a surface pellicle on liquid media.

Two series of inoculations with living cultures of the variant strain were made into guineapigs, one by the intraperitoneal route and the other subcutaneously. These inoculations resulted in transient local suppurations, the intensity of which was increased when a series of injections of sterile culture filtrate was given after the inoculation with

the living fungus. Comparing the results of these inoculations with those of experiments made some years previously with the parent strain, the author concludes that the variant shows slightly increased pathogenicity.

[The animal inoculations did not cause a progressive disease or any condition resembling mycetoma.] *J. T. Duncan.*

ARNOLD, R. & WHILDIN, J. **Rhinosporidiosis of the Conjunctiva. Case Report.** *Amer. Jl. Ophthalm.* 1942, Oct., Ser. 3, v. 25, No. 10, 1227–30, 4 figs.

The author refers to four previous reports of cases of rhinosporidiosis of the conjunctiva, particularly that by ELLES [this *Bulletin*, 1941, v. 38, 724]. In the present case the patient was a white boy, aged 8 years. There was no history of any probable source of the infection. His right eye showed a "pedunculated, lobulated, reddish growth, measuring 6 mm. by 3 mm. by 3 mm. The attachment was in the lateral lower cul-de-sac. . . . The growth was painless."

The tumour was removed under local anaesthesia on April 16th, 1940, and up to February 4th, 1942 (the time of writing) there had been no recurrence.

Microscopical examination showed the typical appearance of the polypus produced by *Rhinosporidium seeberi*; this is described in detail and illustrated by photomicrographs. *J. F. Corson.*

TROPICAL OPHTHALMOLOGY.

A REVIEW OF RECENT ARTICLES. XLII.*

Trachoma.—JULIANELLE[1] reports that he succeeded in grafting trachomatous conjunctival cells on to the chorio-allantois of the developing chick, but failed to obtain any evidence that the virus survived in the newly proliferated cells. Inoculation of these in suitable animals gave negative results. The same observer[2] attempted to adsorb trachoma virus on bacteria in order to demonstrate any agglutination by serum obtained from trachomatous patients and experimentally infected monkeys as well as from artificially immunized monkeys and rabbits, but was unable to obtain any reaction.

The incidence of trachoma amongst children and infants, natives of South Oran, has been studied by FOLEY and PARROT[3] and they report a remarkably high rate of infection; in fact they state that nearly all are attacked before they have reached the age of 12 months. No sign of the disease was apparent, however, in those under the age of three months, and no Halberstaedter-Prowazek bodies could be recovered from their conjunctivae. A single examination sufficed to

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* For the 41st of this Series see Vol. 40, pp. 491–494.

[1] JULIANELLE, L. A. Studies on the Infectivity of Trachoma. XII. Additional Observations on the Cultivability of the Virus. *Amer. J. Ophthalm.* 1943, Mar., v. 26, No. 3, 280–82.

[2] JULIANELLE, L. A. Studies on the Infectivity of Trachoma. XIII. Further Experiments on the Antigenicity of the Virus. *Amer. J. Ophthalm.* 1943, Apr., v. 26, No. 4, 378–81.

[3] FOLEY, H. & PARROT L. Quelques observations sur le trachome des nourrissons indigènes. *Arch. Inst. Pasteur d'Algérie.* 1942, Sept., v. 20, No. 3, 199–203.

demonstrate these bodies in 75 per cent. of older children suffering from the disease and in about one-third of those apparently healthy.

IZAC[4] examined 1,556 Algerians in Laghouat and found trachoma in 81 per cent. of those with fixed abodes, whereas only 31 per cent. of the nomads were infected; some groups of the latter are accustomed to frequent the bazaars and cantonments during winter and spring and among such the incidence was higher. The infectious stage of the disease chiefly affects the juvenile portion of the population; three-quarters are attacked before reaching the age of five and nearly 90 per cent. before the age of 20. Entropion and trichiasis were found present in nearly 6 per cent. of trachomatous patients, the incidence in females being twice as high as in males. Pterygium is much more common among the nomads than among those with fixed habitations, and in this case, owing to the comparatively greater exposure to the influence of hot and dusty winds, the incidence is higher in males than in females.

MITTON and BEATTIE[5] have described their method of exposing the upper fornix to demonstrate the presence of "granules". After eversion of the upper lid the thumb is placed behind the everted tarsus, which is then stretched on the thumb nail. [Study of changes in the upper fornix may be greatly facilitated by the subconjunctival injection of 1 cc. of novocaine solution at the upper edge of the tarsal plate. This procedure enables one to recognize early fibrotic and other pathological changes in the conjunctiva and renders easy the thorough application of remedies.] The authors histologically examined 13 tarsi and 8 lachrymal sacs removed from patients suffering from trachoma in MacCallan's stages III and IV. Generalized thickening of the sac wall together with the development of a lymphoid follicle was observed in one sac. The conjunctival and tarsal changes noted correspond with those found by most other observers. Changes in the Meibomian glands seemed to occur primarily in their ducts. No Halberstaedter-Prowazek bodies and no Rickettsiae were detected in the sections.

BERKLEY[6] recommends an operation for entropion and trichiasis in which the tarsus is divided by an incision through its conjunctival surface. The lid is everted and stretched on a blade of a Desmarres clamp, and a local anaesthetic is injected subconjunctivally, special attention being paid to the angles of the fornix. A horizontal incision, concentric with the lid border and 3 mm. distant from the lid edge, is made through the conjunctiva and the whole thickness of the tarsal plate; care is taken to divide completely the tarsus in its entire width. The cut edges of the tarsus are then undermined on either side of the wound, so as to free it from muscle attachments. One needle of a doubly armed silk suture is next passed through conjunctiva and tarsus at a point 1 mm. behind the centre of the proximal, or upper, lip of the wound and is brought out through the cut surface of the tarsus; it is then passed through the cut surface of the distal tarsal segment and out through the skin just behind the line of the cilia. The second needle is passed through conjunctiva, tarsus, muscle and skin at a point behind and 2 mm. lateral to the site of entry of the first. Four similar mattress sutures are passed, two on either side of the first,

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[4] IZAC, R. Le trachome chez les indigènes de l'annexe de Laghouat. Etude statistique. *Arch. Inst. Pasteur d'Algérie.* 1940, Dec., v. 18, No. 4, 477–91.

[5] MITTON, R. E. R. & BEATTIE, M. K. Diagnosis and Pathology of Trachoma in India. *Indian Med. Gaz.* 1942, Sept., v. 77, No. 9, 517–21, 11 figs. on 1 pl.

[6] BERKLEY, W. L. Treatment of Cicatricial Entropion. *U.S. Nav. Med. Bull.* 1943, May, v. 41, No. 3, 729–36, 2 figs.

and the ends of each are tied over a rubber rod, laid on the lid surface, so as to cause eversion of the edge of the lid. To support the tarsus during the incision and to keep the lid stretched and everted, Berkley uses a Desmarres clamp from which the ring blade has been removed. A suture is passed through the centre of the lid margin; the lid is everted over the remaining blade of the clamp and is held stretched in position by tying the ends of the suture round the setting screw of the instrument.

Cataract.—O'Brien[7] advocates the use of a wide-angle keratome and scissors instead of a Graefe knife in making the section for the extraction of a senile cataract. He claims that the keratome incision is easier to make and results in less astigmatism despite the use of scissors. The scissors cut should be planned to cause a wider wound than is usual with a Graefe knife. [It may prove difficult to ensure an even cut with scissors through the corneo-scleral tissue and some irregularity of the wound is likely to result. This, however, is often less injurious than might be expected.] *H. Kirkpatrick.*

MISCELLANEOUS.

War Medicine. Chicago. 1943, May & June, v. 3, Nos. 5 and 6, 484–97; 619–34, 1 chart. **Development of a Medical Service for Airline Operations in Africa.** [Medical Department, Pan American Airways—Africa, Ltd.]

This paper records the 14 months' experience of the medical department of a major airline running from Bathurst, West Africa, down the coast to Lagos, a distance of 1,731 miles, thence across the continent to Khartoum and Cairo, a total distance of 4,892 miles; later the line was extended to include two routes to India. About a quarter of the line was over the semi-jungle of the coast while inland it traversed more elevated and drier terrain until in the Sudan and Egypt real desert conditions were encountered. Stations were located at intervals of approximately 500 to 700 miles. On the coast temperatures rarely exceeded 90°F., but the humidity was high, the average being 80 per cent., although in parts it did not fall below 90 per cent. for four months at a time. In the desert variations of temperature were greater, the average maximum being 110 to 115°F., but in the Persian Gulf it sometimes reached 127°F.; minimal temperatures in the desert were sometimes as low as 40°F. and diurnal variations of 60 to 80°F. were frequent. Rainfall in parts of the coastal belt reached 200 inches *per annum*, in others only 30 inches and in the desert even less; the rainy season commenced in May, reached a peak in June, and subsided in July with a secondary peak in September.

Applicants for employment were given a complete medical examination including urine analysis, blood counts, serological tests and X-ray examination of the lungs. Of 1,855 persons examined 298 (16·1 per cent.) were rejected, cardiovascular diseases accounting for 20·5 per cent. of the rejections, visual defects for 12·4 per cent., genito-urinary diseases for 11·1 per cent. and respiratory diseases for 10·1 per

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[7] O'Brien, C. S. Comparison of the Keratome-Scissors and Graefe-Knife Incisions for Cataract Extractions. *Amer. J. Ophthalm.* 1943, May, v. 26, No. 5, 508–10.

cent. ; the age limit for service was set at 18–45 years and most of the subjects were below 30. After acceptance each employee was inoculated against smallpox, yellow fever, the enteric fevers, tetanus and cholera, and before sailing was provided with a mosquito net, mosquito boots or leggings, a sun helmet, and a supply of quinine and mosquito repellent. It was recommended that the maximum period of tropical service should be 18 months and that a history of repeated attacks of venereal disease, alcoholism or peptic ulcer should exclude a man from acceptance. Of 1,300 men sent to Africa only 37 returned for medical reasons and none of these was seriously incapacitated. The anticipated hospital bed accommodation was set at 10 per cent. of the personnel but owing to the careful selection of the staff and the control of malaria and gastro-intestinal diseases, 1 per cent. sufficed; it is suggested, therefore, that in a non-combatant area 5 per cent. should be ample provided preventive measures are aggressively applied. It was found that one medical and one surgical officer could care for 600 men, but at each station it was necessary to have a medical officer regardless of the number of the staff ; a dental officer was a necessity and specialists in ophthalmology and nose, throat and ear work were also desirable. Highly qualified laboratory technicians, adequately trained for work in the tropics, were essential, as were sanitarians and a medical entomologist. Emphasis is placed on the proper equipment of hospitals, the provision of a carefully planned laboratory, a dental department and a laundry.

Figures from the military forces furnished by medical authorities with long experience in the area traversed by the airline indicated that the malaria attack rate in West Africa might be 100 per cent. per year, reaching in some places as much as 200 per cent., in fact, during the first few weeks, when the airline was being hurriedly assembled—it was operating in Africa within 60 days of the decision to establish it having been taken in the States—a considerable outbreak of malaria did occur, 46 out of 284 men suffering acute attacks within seven weeks despite daily suppressive quinine, 1,894 man-days of work being lost by 300 men from 1st October to 26th December, 1941. After anti-malaria measures were established the incidence of the disease fell rapidly and cases failed to reappear during the peak of the malaria season ; in September 1942 there was only one case among 1,400 men and in October only three cases among 1,800 men, while comparable organizations in the same area experienced an average monthly rate of 10 per cent. during eight months of the year. The measures adopted were as follows. Each man was provided with an adequate mosquito net. In addition all quarters and accommodation were screened with 18-mesh screening and at each station a maintenance man was provided whose sole duty was to inspect and keep the screening in order. Entrances to buildings were kept at a minimum ; mosquito vestibule traps of adequate length were provided and all doors had automatic spring closing devices. Quarters were sprayed once or twice daily, a trained crew of three men being employed for the purpose, and adjacent native quarters were also sprayed twice weekly. Oiling and dusting of breeding areas were carried out as well as schemes of drainage ; at Accra 45 miles of ditches were constructed at a cost of $16,000. At all sites a maintenance group was constantly available to correct washouts and to prevent potholes from forming. Quinine and atebrin were used for personal suppressive treatment ; there was no evidence that atebrin lowered the ceiling of aviators. Routine blood films were examined of all members of crews immediately before a flight, and of

3,200 smears taken in a year, 35 were found positive for malaria; the value of so examining aviators and of grounding and treating such as were positive was shown by the fact that men with positive smears in practically every instance developed clinical attacks of malaria within 24 to 48 hours. Long sleeved shirts, long trousers and mosquito boots were provided for evening wear. Native quarters were segregated. Various methods of treatment of malaria were tried but there seemed little to choose between them, and a final routine was adopted of quinine sulphate 30 grains daily for two days followed by atebrin 0·3 gm. daily for five days; pamaquin was not used and the relapse rate did not seem unfavourably influenced by its omission. Practically all the malaria was due to *P. falciparum.*

Although clinically not serious, the diarrhoeas ranked next to malaria in importance. Of 600 admissions to the main hospital in eight months, 157 were due to gastro-intestinal diseases of all sorts, and of these 52 occurred in one night from food poisoning, whereas at a large base in the Sudan before adequate sanitary measures were introduced, 20 per cent. of the staff contracted diarrhoea one or more times a month; at one station in Nigeria the rate dropped from 30 per cent. to 0 in one day when properly sanitated quarters and kitchens and flush toilets were installed. Repeated and periodic examination of native food handlers is important since on the basis of 600 stool examinations 28 per cent. of the native population were found to harbour *E. histolytica.* Exclusion of flies from messes and latrines by screening was the most important single factor in the prevention of dysentery.

Infections of the upper respiratory tract were common; of 228 persons confined to quarters during a five month period, 97 had such infections. Fungous infections of the skin were frequent. Psychiatric problems were minimal but all persons were volunteers, living conditions were comfortable and the food was unusually good. There were no special surgical complications among the white persons, sepsis was no more common than in temperate zones and there were no unusual anaesthetic problems; air conditioning of operating theatres was invaluable. At Accra, where the largest hospital was situated, the average daily hospital census from May to December 1942 was 18, the minimum for any month was 9·3 in June and the highest was 25 in November, which was equivalent to 1 per cent., there being 2,500 men in the camp. During the period covered by the report there were eight deaths, and only one of these was due to disease, namely peritonitis following intestinal obstruction. The company drew up a series of health rules and held every man responsible for abiding by them. There was no evidence to indicate that climate *per se* had any deleterious effect on health and it was the unqualified opinion of the medical staff that good health can be maintained in the tropics, despite maximum disease hazard, provided preventive measures are aggressively applied. *F. Murgatroyd.*

WRIGHT, A. A. **Vomiting Sickness in Jamaica.** [Memoranda.] *Brit. Med. J.* 1943, Sept. 25, 392.

The contributor of this note gives brief accounts of two children attacked by the so-called Vomiting Sickness of Jamaica. One, a boy of 12 years, recovered rapidly, the other, a boy of seven years, died in a few hours. [This is the usual history in cases of this disease, but the comments of the author are, to say the least, non-sequential. The

reviewer has repeatedly pointed out the unsuitability of the name "Vomiting Sickness." It is never safe and often misleading to name a disease from one prominent symptom. Dr. Wright mentions ackee poisoning among other causes or possible causes, and of course there is no need to stress that there are several other causes of vomiting and even of fatal disease with vomiting in all countries, tropical and non-tropical, and to limit the name to one disease is no more logical than to call, say, cerebral tumour or meningitis "headache disease." If, however, the definite disease which has gone popularly by this name in Jamaica for more than half a century is separated from others of which this is a symptom, it is better called ackee poisoning. Had the writer studied the many publications on the subject he would not have committed himself to statements such as: "Children eat even the uncooked fruit [ackee] without any untoward results," or that the causation includes "lack of nutritional and vitamin requirements," "exposure to (and shock from) sudden variations in temperature," or, again, "The local authorities have done little, if anything, to probe the cause." We would like to say a few words on each of these, as much danger lies in refusing to recognize the risks of eating ackees under certain conditions.

There is no reason why any untoward results should follow eating of the fruit, cooked or uncooked, provided it is ripe and mature. Nearly 30 years ago SCOTT showed the conditions under which it might be eaten with impunity. Secondly, "Lack of nutrition and vitamins." Is there any condition known where a child can be "well" in the morning and dead in twelve hours from lack of vitamins? Or, if recovery occurs, be quite well again in a day or so without any change from ordinary diet? Thirdly, "exposure to sudden variations in temperature." Jamaica is not subject to great sudden variations in temperature, certainly between say 8 a.m. and 8 p.m. and during that time patients may be attacked and die. Lastly, "the local authorities have done little, if anything, to probe the cause." Apart from the special investigators brought from England by the Jamaica Government and paid for by it, namely T. J. POTTER from London in 1910, and H. SEIDELIN from Liverpool in 1912, each of whom published full reports of his investigations, we need only mention the following: A. MCCATTY, in 1886, R. G. S. BELL, in 1900, H. G. TILLMAN, COOKE, A. THOMSON, G. W. THOMSON, R. S. TURTON who all wrote on the epidemiological and clinical aspects, SCOTT from the epidemiological, clinical, pathological and experimental aspects, based on over 600 cases, the confirmatory work of A. CONNAL and W. RALSTON, the full report of E. O. JORDAN and W. BURROWS, and the admirable experimental work of LEIGH EVANS and ARNOLD. If the writer of the note will read these he will, we feel sure, no longer maintain that the local authorities have done little to probe the cause.] *H. Harold Scott.*

DE FIGUEIREDO, J. Tratamenta das ulceras tropicais pelo vapores de iodo nascente. [**Treatment of Tropical Ulcer by Iodine Vapour.**] *Folha Med.* 1943, May 5, v. 24, No. 9, 81–3.

Reviewing the subject the author notes how common ulcers are in the tropics and how many are the remedies proposed, and divides the latter into three groups: (1) Directed against the local infection—arsenicals, vaccines, pyretotherapy, and surgical as periarterial sympathectomy. (2) Against intercurrent [or rather basal concomitant]

disease, syphilis, helminthiasis, malaria, for example. (3) Directed to improve the general health, iron, strychnine, vitamins. He then describes his method of applying daily "nascent iodine vapour," after cleansing the site with cotton-wool soaked in saline. He spreads on a thick layer of iodoform and by rapid passage of thermocautery liberates the iodine, and then covers with a simple gauze dressing. Another method is by means of an ordinary vaporizer with a rubber bulb.

He quotes briefly ten cases; in one an ulcer of three months' standing had cicatrized after ten applications; in another one of five months' after eight applications; in a third one of 6 months' after nine applications. He suggests the mode of action to be that the nascent iodine on coming into contact with the albuminoid serous secretion of the ulcerated tissues forms a special organic combination which is gradually absorbed and renders conditions unsuitable for the growth of pathogenic organisms, at the same time "raising the defensive power" and stimulating the cells to repair. *H. Harold Scott.*

De, M. N. **Cancrum Oris and Allied Conditions.** *Indian Med. Gaz.* 1943, May, v. 78, No. 5, 245–7, 1 fig.

Noma is, in text-books of medicine, mostly noted as a complication of the infective fevers in children and its existence in tropical conditions is ignored. [It is mentioned, however, in Price's Text-book.] As the author points out, it is by no means uncommon in kala azar and the leishmaniases and in conditions with splenomegaly, because there is "marked depression of leucopoiesis, particularly of granulopoiesis, and . . . blockage of the cells of the reticulo-endothelial system, or . . . a marked inhibition of their antibody-forming function. Both these factors are found to work together in leishmaniasis . . . . The result is that any infective process in the body, however trifling it may be, rapidly progresses and may cause gangrene."

The author refers to cases of splenomegaly characterized by chronicity, development of anaemia, leucopenia, with little or no fever. The gangrene may affect other sites than the mouth, though commonest there because of minor traumas, pyorrhoea and gingivitis. It may involve the cervix uteri, the caecum, the lung and external genitalia. The author calls these noma of the caecum, noma of the lungs, and noma pudendi. [The alternative names he gives, cancrum intestinalis, cancrum pulmonalis are solecisms which cannot be allowed to remain.] Gangrene of the male genitalia has not been previously reported in the literature; the author now records a case in a man of 32 years with kala azar and noma scroti. Rapid improvement followed the use of urea stibamine. *H. Harold Scott.*

Johnson, A. S. **The Pathogenesis of Hepatic Cirrhosis.** *Indian Med. Gaz.* 1943, May, v. 78, No. 5, 227–32, 1 map.

The author describes briefly the diet and habits of the people living in different parts of Travancore. Hepatic cirrhosis has greatest prevalence in the Kottayam area. He then goes on to discuss the reasons for this prevalence and concludes that the cirrhosis can be ascribed to toxins present in their staple food, tapioca. [The whole article is rather an arm-chair discussion and a little positive direct evidence would carry more weight than the many suggestions, probables and possibles, which are put forward. So many things are thought by

the author to play a part ; he considers diabetes, schizophrenia, splenic anaemia, degeneration of the lenticular nucleus, manganese poisoning, malaria, alcohol, curries, and others, all in four pages.] In parts where this tapioca is used, but cirrhosis is not common, vitamin disturbance is adduced. Alcohol, he maintains, does not produce cirrhosis though it may act indirectly by setting up gastro-enteritis and permitting absorption of poisons. Neurasthenia and flushings " indicate the onset of cirrhotic change in the liver." This " may be due to the liver cells liberating histamine, as they are being subjected to the action of toxins."

The discussion indicates that the author has a wide field for research open to him and the following up of some of his surmises may throw much needed light on this difficult subject. *H. Harold Scott.*

KELLNER, F. " Flüchtiges Lungeninfiltrat "—" eosinophile Pneumonie." [**Transient Infiltration of the Lungs—" Eosinophil Pneumonia."**] *Münch. med. Woch.* 1943, Apr. 23, v. 90, No. 16/17, 287–8.

This is not a discussion of the condition itself, but of the correctness of the terms used. The author traces the history of our knowledge of the disease from the time when, after the last war, cases of " infiltrative disease of the lungs " began to be met with in increasing numbers. Some ten years ago the presence of eosinophilia was observed as a common accompaniment of the lung condition and the term " infiltration with eosinophilia " was applied to it. Next, the word " infiltration " was inevitably associated with tuberculosis and when this organism was not found the adjective " non-specific " was added. Eosinophilia is common to many diseases and in this condition is subject to much variation, hence neither " eosinophilic " nor " infiltration " was altogether satisfactory. Yet further, " inflammation of the lungs " to many, perhaps most, physicians implied pneumonia, and the very transitoriness of the condition ruled this out, although the infiltration was both intra- and inter-alveolar. The idea that it might be due to the passage of larval Ascaris was mooted, but, though this might account for some cases, it certainly left many unexplained. The author maintains that the term " infiltration " should be reserved for tuberculous lesions ; on the other hand the condition is pneumonic, but to assign the name " eosinophil pneumonia " to it is, he says, premature. He seems to favour the view of VON MEYENBURG that this transient infiltration is allergic in nature and the eosinophilia when present supports the idea. [This article is an interesting review of the question in spite of the fact that no very tangible conclusion is reached. See also this *Bulletin*, 1943, v. 40, pp. 720, 721.]

H. Harold Scott.

MURAZ, G. Koumra (Afrique Équatoriale Française) et ses 80 pour 100 de goitreux. [**Koumra and its 80 per cent. of Goitrous Inhabitants.**] *Presse Méd.* 1943, June 26, v. 51, No. 24, 349–50, 4 figs. (1 map).

Referring to a recent paper by Robert DUPONT (*La Presse Médicale*, 1941, Jan., Nos. 5–6), on goitre in Koumra, in the Middle Chari area of French Equatorial Africa, the author points out that the disease in this area was first studied in 1917 by BOUILLEZ while directing measures

against sleeping sickness. The author, who succeeded Bouillez, again investigated the problem of the frequency of goitre in this area in 1922. It affected the population of the laterite [iron-stone] plateau who got their water from deep wells, while the riverine people were free from the disease. The latter, however, were infected with sleeping sickness, while the people of the plateau escaped this disease, and the author was faced with the problem of how to deal with these two populations, as neither could safely be transferred to the other area. He advised the distribution of iodized salt, but transport difficulties prevented the plan from being carried out. Failing this he considers that surgical removal of nodular goitres, now being done by DUPONT for the first time in this region, is a valuable measure which will attract many sick people to the hospital and add to the renown of French science. *J. F. Corson.*

CIMMINO, V. Studio sulla velocità di sedimentazione in Eritrea. Nota 1.—Considerazioni generali—Valori normali. [**Blood Sedimentation Rate in Eritrea; Normal Values.**] *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1943, v. 2, No. 2, 10–16. [21 refs.] English summary.

" The author studies the speed of sedimentation in the more common diseases of Eritrea, starting the work with research on healthy subjects, either European or Native. After having summarised the various methods used for this test and the various causes which can influence the speed, he describes the macromethod used by him, viz.: that of KAUFMANN. He reports the normal values obtained by this method.

" Once more he calls the attention of the student to the importance which various factors may assume in the estimation of this interesting biological phenomenon, which is still worth further study and needs greater accuracy."

CIMMINO, V. Studio sulla velocità di sedimentazione in Eritrea. Nota 2. La V. di S. nella tubercolosi pulmonare e nella lues. *Boll. d. Soc. Italiana di Med. e Igiene Trop.* (Sez. Eritrea.) Asmara. 1943, v. 2, No. 2, 55–63. [14 refs.] Nota. 3. La V. di S. nel tifo esantematico. *Ibid.* 64–70. Nota 4. La velocità di sedimentazione nella malaria. *Ibid.* 71–5. Nota 5. La velocità di sedimentazione nella febbre ricorrente. [**The Blood Sedimentations in Various Diseases of the Natives of Eritrea.**] *Ibid.* 76–82. English summaries.

" 1. Pulmonary tuberculosis in Europeans living in the Eritrean highland, modifies the speed of sedimentation in just the same proportion as that described in Europe (it is generally increased in the active forms and vice-versa).

" 2. Pulmonary tuberculosis in natives is very often accompanied, and in our cases was always accompanied, by a great increase in the speed of sedimentation, thus confirming the acute tendency of the tubercular process in such patients.

" 3. Active untreated syphilis in Eritrean natives constantly accelerates the speed of sedimentation.

" 4. Syphilis in natives, resistant to treatment, is not accompanied by any slowing of the speed of sedimentation; on the contrary, in general the speed is accelerated, as in untreated syphilis."

"In both European and native patients, during the 2nd week of typhus, there is an increase in the speed of sedimentation. During convalescence, readings slowly return to normal; this return to normal is less evident in natives. There is no relation between the gravity of the illness, the intensity of the Weil-Felix reaction and the speed of sedimentation."

"While in Europeans, malaria has little influence on the speed of sedimentation (or at least shows an inconstant effect) in Eritrean natives, the infection is almost constantly accompanied by a perceptible increase in the speed."

"Relapsing fever produces an increase in the speed of sedimentation, during both the period of fever and the apyrexic periods.

"Specific therapy, producing cure of the disease, does not bring back to normal readings the speed of sedimentation. This is because of the fact that although the action of the spirochete has ceased, there remains for a long time the accelerating action of arsenobenzol preparations.

"The experiment seems of little value therefore, for the study of relapsing fever."

BRICEÑO-IRAGORRY, L. Nota sobre simulidos del Valle de Caracas. [**Simuliidae of the Valley of Caracas.**] *Bol. Laboratorio Clinica "Luis Razetti."* Caracas. 1943, May, v. 3, No. 10, 181–6, 4 figs.

HINTON, H. E. & CORBET, A. S. **Common Insect Pests of Stored Food Products. A Guide to their Identification.** *British Museum (Natural History) Economic Ser. No. 15.* 1943, June, 44 pp., 87 figs. London: Printed by Order of the Trustees of the British Museum. [1s.] [Summary appears also in *Bulletin of Hygiene.*]

Of the vast numbers of insect species known, relatively few have become pests of stored food products. But such as have been able to adapt themselves to the ecological conditions of food stores, take advantage of the almost unlimited food supply to become serious pests. Trouble from these pests is especially prevalent now when much food is being stored both on a wholesale and on a domestic scale.

This British Museum publication is a guide to the identification of the more common food pests. It will prove an invaluable *vade-mecum* to the inspectors of the Ministry of Food, Pest Infestation Branch. And, since pests in domestic stores and larders are often sent to the local Health Department, it will also be useful to Sanitary Inspectors.

The bulk of pests of stored food belong to the orders Coleoptera and Lepidoptera, and these are considered in greatest detail. The principal families of beetles are distinguished by a key and under each family a separate key traces the 51 species mentioned. The section on moths distinguishes between the groups Pryalidina and Tinaeina and gives a key to 19 pest species contained in them.

The diagnostic characters are carefully explained and illustrated by line drawings. *J. R. Busvine.*

COWEN, D., WOLF, A. & PAIGE, Beryl H. **Toxoplasmic Encephalomyelitis. VI. Clinical Diagnosis of Infantile or Congenital Toxoplasmosis; Survival beyond Infancy.** *Arch. Neurol. & Psychiat.* 1942, Nov., v. 48, No. 5, 689–739, 9 figs. [Refs. in footnotes.]

In this paper the authors review the symptoms in nine cases of infantile or congenital toxoplasmic encephalomyelitis which were

diagnosed at necropsy by the discovery of the parasite. The children all died during infancy in the acute or subacute stage of the infection. The outstanding features were the occurrence of striking ocular lesions associated with neurological symptoms and signs. The ocular lesions consisted of multiple focal bilateral areas of chorio-retinitis, in most cases involving the macula, with less constant microphthalmos, nystagmus and ocular palsies. The neurological signs included convulsions, hydrocephalus and, most strikingly, multiple foci of intracerebral calcification. On the basis of the nine fatal cases it was possible to construct a picture which enabled six cases to be diagnosed clinically. These were in older children, all alive at the time of writing, in whom the duration of the disease was from 14 months to 8 years, showing that the infection is not uniformly fatal and that it may become chronic, healed or latent. In these older children the outstanding symptom is usually diminution in vision due to the effect of the multiple foci of healed chorio-retinitis; strabismus, microphthalmos and minor congenital ocular defects may also be present. Generalized convulsions or petit mal attacks may persist or make their appearance. Internal hydrocephalus may become chronic and progressive. Foci of intracerebral calcification persists and may increase in number and size. Retardation in the development of speech and minor degrees of mental deficiency occur. It would seem that in all these cases the infection is contracted before birth and that the form of disease should be regarded as infantile or congenital. In other cases toxoplasmosis is not acquired till the juvenile or adult period of life is reached. In such cases the infection is termed juvenile or adult acquired toxoplasmosis.

It is evident that congenital toxoplasmosis is not a rare disease and it is believed that many cases have been erroneously regarded as instances of congenital malformation of the brain, cerebral birth injury, epilepsy, congenital hydrocephalus, etc. The identification of other cases of the disease, which is evidently widespread in the United States, may yield some information as to its epidemiology.

C. M. Wenyon.

BOOK REVIEWS.

CAMBOURNAC, Francisco José C. **Sôbre a epidemiologia do sezonismo em Portugal.** [On the Epidemiology of Malaria in Portugal.] —235 pp. With 44 graphs. 1942. Lisbon: Sociedade Industrial de Tipografia, Lda. R. Almirante Pessanha 5.

This is a very detailed account of the epidemiology of malaria in Portugal and is admirably documented. The geographical distribution of the disease in the country and the factors responsible for that patchy distribution are discussed at length. The chapter dealing with the Anopheles of Portugal, *A. nigripes*, *A. claviger*, and *A. maculipennis* vars. *typicus* and *atroparvus*, is very complete. *A. maculipennis* var. *atroparvus* is by far the most important vector of malaria in Portugal.

The author is pre-eminently well qualified for the task he has so successfully accomplished. Work in the antimalaria stations in

Benavente and Alcácera do Sol was followed by many years' collaboration with Rolla B. HILL in the comprehensive investigations that were sponsored by the General Directorate of Public Health of Portugal and the International Health Division of the Rockefeller Foundation. In 1938 the intervention of the Rockefeller Foundation culminated in the creation of the Malariological Institute at Aguas de Moura, which is now the centre of malaria studies for the whole country. Dr. Cambournac became the Director of the Institute.

Most of the author's more important observations regarding the biology of *A. maculipennis* var. *atroparvus* in Portugal and the association of malaria with rice cultivation have previously been published in medical journals and have been summarized from time to time in this *Bulletin**. Their re-statement and expansion in this very complete study of the epidemiology of malaria in Portugal constitute a noteworthy addition to malaria literature. *Norman White.*

CLAVERO DEL CAMPO, G. [Director del Instituto Nacional de Sanidad] & PÉREZ GALLARDO, F. [Médico de Sanidad Nacional]. **Técnicas de laboratorio en el tifus exantemático.** Prólogo del Prof. Dr. J. A. PALANCA. [Laboratory Technique in Typhus Exanthematicus.] —185 pp. With 104 figs. (7 coloured). 1943. Madrid: Imprenta de Prensa Española, S. A. Serrano 61.

A book by the same authors on louse-borne typhus fever has already been reviewed [this *Bulletin*, v. 40, 531.] The present small volume of 185 pages deserves equal commendation.

All the more important methods employed in the investigation of louse-borne and flea-borne typhus are clearly described, and instructions are given for the preparation of killed vaccines. Special attention has been paid to the pitfalls and fallacies connected with the various procedures. There are 104 illustrations, of which seven are coloured; these have been specially prepared for the book, they are exceptionally clear and self-explanatory so that they will be found very helpful by all who undertake laboratory investigations into the Rickettsial fevers.

The authors deal only with methods of which they have had personal experience, and, therefore, there are certain omissions, such as the preparation of living vaccines and the technique of the complement fixation test. The methods of isolating and cultivating Rickettsiae by guineapig inoculation, intrarectal inoculation of lice, and yolk-sac cultures, occupy about 88 pages. Murine Rickettsiae are dealt with in a further section of 28 pages, though these are said never to have been isolated in natural conditions in Spain. The Weil-Felix and Rickettsia-agglutination reactions are described but no comment is made regarding their relative merits. The description of the latter reaction is based on the use of Rickettsial suspensions prepared from laboratory-infected lice. Giroud's serum-protection test is well described.

The rest of the book deals with the preparation of vaccines by Weigl's, Cox's, and Durand and Giroud's methods, all of which are described in detail and with a wealth of illustration.

Some workers will think that undue attention has been paid to Weigl's vaccine, which the authors obviously regard as having special advantages over the others. There must be few who can contemplate

* 1937, v. 34, 144; 1939, v. 36, 920; 1940, v. 37, 784; 1941, v. 38, 564.

undertaking the elaborate louse-husbandry procedures involved in the preparation of Weigl's vaccine.

Nothing is said about the risks run by the personnel engaged in the preparation of vaccines.

The authors state that the Weil-Felix reaction rapidly became negative after attacks in the recent Spanish epidemic. Titres higher than 1–25 to 1–50 two months after attacks were of rare occurrence.

Kudicke and Steuer's "dry-blood agglutination test" is praised, but it is said that positive results can be expected only when the Weil-Felix titre is higher than 1–200.

In the opinion of the authors the egg-yolk and rodent-lung vaccines have not yet been proved to be effective for human beings; their dosage has not yet been established; doses suitable for guineapigs are not necessarily adequate for human beings. In this matter the authors express doubts which must have occurred to many others, but the same misgivings apply also to Weigl's vaccine, and if much larger doses are found to be necessary the cost of this will be prohibitive.

The Director General of Public Health, Prof. PALANCA, has written a prologue, in which he gives an interesting account of the work of his department during the recent epidemic. He mentions that the book was prepared on his initiative and published at the expense of the Health Department. *John W. D. Megaw.*

SHATTUCK (George Cheever) [M.D.] & MIXTER (William Jason) [M.D.]. **Handbook of Health for Overseas Service.** pp. vii+228. With 15 figs. Second Edition, Revised. 1943. Cambridge, Mass., Harvard University Press.

As stated in the preface, this second edition has been extensively revised and new material has been added. It is intended for the use of persons who may be in places where hygienic conditions are primitive and medical help at times unobtainable, and it admirably fulfils that purpose. The chapters include general directions for personal hygiene and sensible living, and an account of common ailments and medical and surgical affections—including the more important tropical diseases. Directions for the use of sulphonamide compounds are given, with a warning of the risks involved. There is a short chapter on the maintenance of health in arctic climates. The chapter on surgery deals with the commoner surgical emergencies, including such recently important conditions as immersion foot and lifeboat foot. A short chapter on miscellaneous medical information contains directions for the use of a thermometer, hypodermic and antitoxin syringes and the more important drugs.

The book is of pocket size and well printed with water-resisting ink on water-resisting paper. It can be strongly recommended to those for whose use it was written. *J. F. Corson.*

STRONG (Richard P.). [M.D., Sc.D., D.S.M., C.B., Professor of Tropical Medicine, Emeritus, Harvard University, etc.]. **Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases.** Sixth Edition in 2 Volumes. Vol. I. pp. xv+1–871+xl. Vol. II. pp. vii+872–1747+xl. With 398 figs. & 4 plates (2 coloured). Reprint with additions. 1943. Philadelphia: The Blakiston Company. [Price £5 5*s*.]

As predicted, the demand for the Sixth Edition [this *Bulletin*, 1942, Vol. 39, p. 352] has rapidly exhausted the issue so that a reprint has

become necessary. The pagination remains as before, but here and there additional paragraphs and alphabetically numbered supernumerary pages have been wedged in to accommodate recent information.

In treatment of leishmaniasis stilbamidine finds a place; in yellow fever there is a considerable addendum, and also in trench fever and sprue. It is rather unfortunate that on p. 1332 it is suggested that *Wuchereria malayi* might be identical with the Pacific variety of *W. bancrofti.*

The confidence of the profession in this excellent book is reflected in this up-to-date reprint which has been effected in such a short space of time. *P. Manson-Bahr.*

SIMMONS, J. S. & AITKEN, T. H. G. **The Anopheline Mosquitoes of the Northern Half of the Western Hemisphere and of the Philippine Islands (Distribution, Habits, Identification, Importance as Vectors, and Control).** *Army Med. Bull. No. 59.* (Special Issue). 1942, Jan., 213 pp., 5 pls. & 2 charts. [19 pages of refs.] Pennsylvania: Medical Field Service School, Carlisle Barracks.

The authors' purpose has been to gather into one small volume facts on the *Anopheles* mosquitoes of the rather heterogeneous regions defined in the title. The book is for the use of military medical men, and entomologists in the U.S. Army Sanitary Corps.

The authors deal successively with three areas, North America, Tropical America and the Philippine Islands: for each they give keys for adult mosquitoes of both sexes, and for larvae. These are followed by short notes on geographical distribution, biology and relation to malaria of each species. The evidence on relation to malaria is tabulated fully.

There is a rather brief chapter on malaria surveys and control, and appendices on laboratory methods. The list of references is full.

The book appears to fulfil its purpose of tabulating and presenting necessary facts. To the British reader the section dealing with Tropical America (including the Islands) is likely to be valuable, for the fauna of that area is complex (40 *Anopheles* are listed), knowledge has recently increased, and collective works are few.

P. A. Buxton.

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INDEX OF AUTHORS OR SOURCES.

The bracketed abbreviations after the page numbers indicate the subjects. Page numbers within brackets indicate papers not summarized.

Am. signifies Amoebiasis and Intestinal Protozoal Infections.
Bart. ,, Bartonellosis
Bb. ,, Beriberi.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
Der. ,, Dermatology and Fungous Diseases.
Diet. ,, Deficiency Diseases including Epidemic Dropsy.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers, including Dengue and Sandfly Fever.
Haem. ,, Haematology.
Hel. ,, Helminthiasis.
Leish. signifies Leishmaniasis.
Lep. ,, Leprosy.
Lept. ,, Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Oph. ,, Tropical Ophthalmology.
Pl. ,, Plague.
Rab. ,, Rabies.
R.F. ,, Relapsing Fever and other Spirochaetoses.
Sp. ,, Sprue.
Spec. Res. signifies Special Research.
Tryp. signifies Trypanosomiasis.
Typh. ,, Typhus.
Vms. ,, Venoms and Antivenenes.
Y.F. ,, Yellow Fever.
Y. & S. ,, Yaws and Syphilis.

A.

Abicht, I. & Kuhlmann, F., 937 (Sp.)
Abonnenc, E., with Senevet, (870) (Misc.)
——, with —— & Chabelard, (870) (Misc.)
Abrikosov, A. I., 764 (Typh.)
Achundov, J. A., with Popov, 314 (R.F.)
Adler, S. & Ashbel, R., 55 (R.F.)
Adlersberg, D. & Sobotka, H., 621 (Sp.)
Afanassiev, S. F., 521 (Mal.)
Aguiar, J., with Fox, Lennette & Manso, 44 (Y.F.)
Aguilar Meza, R., González, E. & Medrano, A. R., 292 (Mal.)
Aguirre Pequeño, E., 330 (Der.)
Ahmad, D., 181 (Misc.)
Ahmed, N., 219 (Mal.), 259 (Diet.)
Ahrens, W., 529, 600 (Typh.)
Aitken, T. H. G., (113) (Mal.)
——, with Simmons, 954 (B.R.)
Alameda County Mosquito Abatement District Annual Report 1942, (636) (Misc.)
Albarracín, L., 549 (Lep.)
Alberto Alvarado, C., 745 (Mal.), 773 (Pl.)
Alcay, L., Marill, F. G. & Musso, J. C., 467 (Hel.)
——, with —— & ——, 466 (Hel.)
——, with Senevet, Moutier & Bourgarel, 805 (Misc.)
Alemany Vall, R. & Castells Escuder, A., 176 (Der.)
Alessandri, H., Palazuelos, P. G. & Lerner, J., 168 (Diet.)
Alfonso y Armenteros, J., with Castro Palomino, 630 (Der.)
Alicata, J. E. & Breaks, V., 767 (Typh.)
d'Allaines, F., Lavier, G. & Gandrille, 472 (Hel.)
Allen, P. H. 724 (B.R.)
Alonso Mujica, J. C., 774 (Pl.)
Alpins, O., 629 (Der.)
Altmann, A., with Foy, Barnes & Kondi, 363 (Bl.)
Alvarado, C. A., 272 (B.R.)
Alvayay, J. & Carvajal, V., 761 (Tryp.)
Alwall, N., 259 (Diet.)
Alwens, W., 687 (Typh.)
Amaral, J., 289 (Mal.)
d'Amato, H. J., 219 (Mal.)
Ambialet, R., with Parrot & Catanei, 739 (Mal.)
Amolsch, A. L., with Palmer & Shaffer, 330 (Der.)
Anderson, L.A.P., with Swaminath & Shortt, 227 (Leish.)
Anderson, T. F., with Plotz, Smadel & Chambers, 685 (Typh.)
Andeze, P. J., with Potenza, 891 (Leish.)
Andrews, J., 615 (Hel.)
——, with Hill, 324 (Hel.)
——, Howard, Jr. & Turner, 673 (Mal.)
——, with Howard, R. S., Jr., 16 (Mal.)
Andrews, J. C. & Webb, B. D., 158 (Hel.)
Anduze, P. J., 537 (Y.F.)
Anigstein, L. & Bader, M. N., 386 (Fev.)
d'Antoni, J. S., 53, 697 (Am.)
Arce, J., (66) (Hel.)
Arguedas Klée, G., with Cofiño Ubico, 712 (Diet.)
Arneth, J., 301 (Fev.)
Arnold, H. L., Jr., 175 (Der.)
Arnold, R. & Whildin, J., 492 (Oph.) 941 (Der.)
Ashbel, R., 55 (R.F.)
——, with Adler, 55 (R.F.)
Ashour, M., with Awad, (323) (Hel.)
Assratian, S. N., (81) Vms.)
Augustine, D. L., 758 (Tryp.)
Austin, C. J., 315, 700 (Lep.)
Austin, T., 397 (Lep.)

Avendaño, O., with Palacios, 659 (Rab.)
Avery, H., (378) (Leish.)
Awad, S. H. & Ashour, M., (323) (Hel.)
Axtmayer, J. H., with Krakower & Hoffman, 923 (Hel.)
——, with —— & Morales-Otero, 789 (Lep.)
Ayala, F. & Bravo, G., 751 (Mal.)
Aykroyd, W. R., 935 (Diet.)
—— & Verma, O. P., 491 (Oph.)
Ayyar, P. V. S., with Menon & Shortt, 220 (Mal.)
Azúa, L., with Gay Prieto, López de Azcona, Bondeu & Cárdenas, 94 (Misc.)

B.

Bacigalupo, J., (65) (Hel.)
Bader, M. N., with Anigstein, 386 (Fev.)
v. Baeyer, W., 831 (Typh.)
Bailly, J., with Remlinger, 195, 196, 197, 198, 199, 653 *bis* (Rab.)
Baker, G. E., 902 (Typh.)
Baker, R., with McDermott, Webster, Lockhart & Tompsett, 679 (Tryp.)
Baltazard, M., with Blanc, 85 (Misc.)
Banerjee, D. N., 454 (Chl.)
Bang, F. & Simpson, T., 359 (Mal.)
Bang, F. B., 206 (Rab.)
——, Quinby, G. E. & Simpson, T. W., 817 (Mal.)
Barbará, L., with Toranzos & Figuerero, 680 (Tryp.)
Barber, H. J., Slack, R. & Wien, R., 376 (Leish.)
Barber, J. F., 822 (Mal.)
Bargen, J. A., with Berkman, 243 (Am.)
Barker, P. B., with Donald, 30 (Typh.)
Barlovatz, A., 101 (Mal.), 327 (Hel.)
—— Joukovsky, T., 37 (Fev.)
Barnes, H. D., with Foy, Altmann & Kondi, 363 (Bl.)
Barnéoud, J. & Gaud, J., 469 (Hel.)
Barnetson, J., with Simson, 176 (Der.)
——, with —— & Harington, 630 (Der.)
Barnett, L., (926) (Hel.)
Baron, B. & Brunner, M., 477 (Hel.)
Barreras, L., with Cabrera Calderin & Scull., 262 (Hel.)
Barreto, J. de B., 182 (Misc.)
Barth, C., 134 (Typh.)
Barwell, C., with Grant, 498 (Misc.)
Basso, G., with Mazza & Basso, R., 22, 226 (Tryp.)
Basso, R., with——& Basso, G., 22, 226 (Tryp.)
Basu, A. K., 717 (Misc.)
Basu, C. C., with Brahmachari, 121 (Leish.)
Bates, M., 804 (Misc.)
Baumann, H., with Schwetz, 103, 211 *bis*, 212 (Mal.)
——, with ——, Beumer, Mme. & Fort, 213, 357 (Mal.)
——, with —— & Fort, 743 (Mal.)
Bearcroft, W. G. C., with van Rooyen, 895 (Typh.)
Beasley, B. T., 939 (Vms.)
Beattie, M. K., with Mitton, 942 (Oph.)
Becker, B. J. P., 562 (Der.)
Becmeur, A., 708 (Hel.)
de Beer, E. J., with Bercovitz & Page, 929 (Hel.)
Beerman, H., 629 (Der.)
Béguet, M. & Horrenberger, R., 657 (Rab.)
Beklemishev, W. N., 511 (Mal.)
Belding, D. L., 271 (B.R.)
Bellamy, R. E., 215 (Mal.)
Beltrán, E., 455, 460, (782) (Am.)
—— & Bustamante, M. E., 441 (Leish.)
—— & Lopez Portillo, S., 459 (Am.)
Benhamou, E., 893 (Typh.)
Bentz, with Fain, 218 (Mal.)
Bercovitz, Z., (495) (Misc.)
——, Page, R. C. & de Beer, E. J., 929 (Hel.)
Berkley, W. L., 942 (Oph.)
Berkman, J. M. & Bargen, J. A., 243 (Am.)
Berkman, S., 538 (Pl.)
Berman, L. S., with Hermann, 457 (Am.)
Berne, C. J., 146 (Am.)
Bernkopf, H., with Kligler, 651 (Rab.)
Berry, W. T. C., 716 (Misc.)
Beumer, Mme., with Schwetz, Baumann & Fort, 213, 357 (Mal.)
Bevier, G., 512 (Mal.)
Bhave, P. D. & Bopaiya, M. S., 170 (Haem.)
Bibikova, T. I., 143 (Dys.)
Bickhardt, R., with Otto, 129 (Typh.)
Bigham, J. T., 339 (Misc.)
Biraud, Y., 378 *bis* (Typh.)
Bird, R. M., with Kauer & Reznikoff, 560 (Vms.)
Birks, P. H., 749 (Mal.)
Bishop, A., 17, 583 (Mal.)
Bishop, L. K., with Eyles, 667, 817 (Mal.)
Biswas, T. C., with Roy, 112 (Mal.)
Bittencourt, A., with Penna, 840 (Y.F.)
Blacklock, D. B., (85), 631 (Misc.)
—— & Wilson, C., 358 *bis* (Mal.)
Blakhov, A. A. & Kuptzova, A. D., 435 (Mal.)
Blanc, G. & Baltazard, M., 85 (Misc.)
Bloch, E. H., with Knisely, 584 (Mal.)
Blomfield, D. M., 627 (Der.)
Blondel, P., with Poirier, 848 (Hel.)
Bloss, J. F. E., 633 (Misc.)
Blum, B. B. & Lilga, H. V., 552 (Hel.)
Boase, A. J., 177 (Der.)
Bobrov, M. V., 441 (Leish.)
Boehnhardt, H., 839 (Fev.)
Boehrer, J. J., Stanford, C. E. & Ryan, E., 619 (Diet.)
Bogendörfer, 298 (Typh.)
Boix Barrios, J., 23 (Leish.)
Boland, E. R., 540 (Dys.)
Boletim do Serviço de Framboézia, 546 (Y. & S.)
Boletin de la Oficina Sanitaria Panamericana, 487 (Der.)
Boletin Sanitário, 774 (Pl.)
Bolten, J., 753 (Mal.)
Bondeu, F., with Gay Prieto, López de Azcona, Azúa & Cárdenas, 94 (Misc.)
Bonne, C., 325, 553 (Hel.)
Bopaiya, M. S., with Bhave, 170 (Haem.)
Boquet, P. & Vendrely, R., 862 (Vms.)
v. Bormann, F., 838 (Fev.)
Boulnois, J., 741 (Mal.)
Bourgarel, R., with Senevet, Moutier & Gros, 805 (Misc.)
Bourguignon, G. C., 103 (Mal.)

Boyd, G. H. & Gilkerson, S. W., 221 (Mal.)
Boyd, J. S. K., with Fairley, 540 (Dys.)
Boyd, M. F., 11, 751 (Mal.)
—— & Kitchen, S. F., 818 (Mal.)
Boyer, F., with Nitti & Conge, 57 (R.F.).
Bozicevich, J., with Rees, Reardon & Jones, 456 (Am.)
——, with Wright, (854) (Hel.)
Brady, F. J., Jones, M. F. & Newton, W. L., 782 (Am.)
Brahmachari U., 417 (B.R.)
—— & Basu, C. C., 121 (Leish.)
Braune, J. F., 252 (Hel.)
Bravo, G., with Ayala, 751 (Mal.)
Brazzaville [Afrique Francaise Libre], 833, 887 (Tryp.), 907 (Y.F.)
Brea, M. M., Niño, F. L. & Taiana, J. A., 698 (Am.)
Breaks, V., with Alicata, 767 (Typh.)
Brewer, A. E., 308 (Dys.)
Briceño-Iragorri, L., 51 (Dys.), (950) (Misc.)
Briceño Rossi, A. L., 845 (Y. & S.)
Brigham, G. D. & Pickens, E. G., (690) (Typh.)
—— & Watt, J., 238 (Fev.)
Brighenti, D., 580 (Mal.)
Brill, N. Q. & Pellicano, V. L., 819 (Mal.)
British Empire Leprosy Relief Association (Indian Council), 57 (Lep.)
British Empire Leprosy Relief Association (Madras Provincial Council), 397 (Lep.)
British Guiana, 512 (Mal.)
Britton, C. J. C., with Whitby, 503 (B.R.)
Brocklebank, J. A., 868 (Misc.)
Broders, A. C., Dochat, G. R., Herrell, W. E. & Vaughn, L. D., 798 (Der.)
Brodie, B. B. & Udenfriend, S., 821 (Mal.)
Brody, A. L. & Knipling, E. F., 636 (Misc.)
Bronshteín, N. I. & Zak, O. S., 661 (Rab.)
Brown, H. E., with Derrick & Smith, 302 (Fev.)
Browne, G. St. J. O., 866 (Misc.)
Brulé, M. & Pestel, M., 722 (Misc.)
Brumpt, E. & Brumpt, L.-C., 302 (Fev.)
—— & Dao Van Ty, (359) (Mal.)
Brumstein, M. S., with Mogilnizky, 141 (Dys.)
von Brunn, E., with Hoff, 299 (Typh.)
Brunner, M., with Baron, 477 (Hel.)
Bruynseels, G., with Dubois, (176) (Der.)
Btesh, S., with Reitler, 333 (Misc.)
Buch, E., with Kunert, 130 (Typh.)
Bucholz, D. J., with Marble & Skoog, 258 (Hel.)
Bulletin de l'Institut d'Hygiéne du Maroc, (563) (Misc.), 604 (Pl.)
Bulmer, E. & Priest, W. M., 395, 777 (Dys.)
Burga Saavedra, V., 306 (Pl.)
Burkitt, R. W., 677 (Mal.)
Burkwall, H. F., 827 (Bl.)
Burnet, F. M., 234 (Fev.)
Burruss, H. W., with Hargett & Donovan 692 (Y.F.)
Burschkies, K., 610 (Lep.)
Burt, J. C., Lane, C. M. & Hamilton, J. L., 923 (Hel.)
Burtt, E., 369 (Tryp.)
Bury, K. J., 31 (Typh.)
Bustamante, M. E., 388 (Y.F.)
——, with Beltran, 441 (Leish.)
Bustamante, M. E., Kumm, H. W. & Herrera, J. R., 453 (Y.F.)
——, with —— & ——, (878) (Mal.)
Busvine, J. R., 601 (Typh.)
Byrd, E. E., 65 (Hel.)

C.

Cable, R. M., with Headlee, 148 (Am.)
Cabral, A. S., with Fox, 451 (Y.F.)
Cabrera Calderín, J. G., Scull, J. M. L. & Barreras, L., 262 (Haem.)
Caldwell, F. E. & György, P., 891 (Tryp.)
Caldwell, G. T., 411 (Der.)
Callaway, J. L., with Noojin, 797 (Der.)
Cambournac, F. J. C., 951 (B.R.)
Cambournac, with Hill & Simões, 818 (Mal.)
Cameron, J. A. M., 916 (Am.)
—— & Collins, J. C., 144 (Am.)
Cameron, J. D. S. & Lawler, N. A., 458 (Am.)
Campbell, D. H., (554) (Hel.)
——, with Melcher, 328 (Hel.)
Campbell-Begg, R., 550 (Hel.)
Campourcy, 18 (Tryp.)
Canas, E., with Volk, 410 (Der.)
Cannon, D. A., 415 (Misc.)
Cantrell, W., with Huff & Coulston, 815 (Mal.)
Cárdenas, M., with Gay prieto, López de Azcona, Azúa & Bondeu, 94 (Misc.)
Carlé with Tietz, 133 (Typh.)
Carman, J. A., 518 (Mal.)
Carpenter, S. J., (413) (Misc.)
Carr, H. P. & Hill, R. B., 745 (Mal.)
Carrión, A. L., 410 (Der.)
Carretero, A. V., 36 (Typh.)
Carvajal, V., with Alvayay, 761 (Tryp.)
——, with Gasic, 760 (Tryp.)
Casals, J., with Webster, 201 *bis* (Rab.)
Casini, G., 286 (Mal.)
Casiraghi, J. C., with Pirosky, I. & Pirosky, R. de, 474 (Hel.)
Cassinelli, J. F., with Lasnier, 614 (Hel.)
Castañeda, M. R., 127, 128 (Typh.)
Castañe Decoud, A., 547 (Lep.)
Castells Escuder, A., with Alemany Vall, 176 (Der.)
Castro Palomino, J. & Alfonso y Armenteros, J., 630 (Der.)
Catanei, A., 940 (Der.)
——, with Parrot & Ambialet, 739 (Mal.)
Cattapan, A., with Cupi, 124 (Leish.)
Causey, O. R., 579 (Mal.)
——, Deane, L. M. & Deane, M. P., 580 (Mal.)
——, with Deane, M. P., 581 (Mal.)
——, Penido, H. M. & Deane, L. M., 815 (Mal.)
Cavazzi, G., 901 (Typh.)
Cawston, F. G., 553 (Hel.)
Ceccaldi, J., 833 (Typh.), 887 (Tryp.), 907 (Y.F.)
—— & Guilhaumou, F., 56 (R.F.)
Celis Pérez, A., 715 (Der.)
Ceylon, 854 (Diet.)
Chabelard, R., (433) (Mal.)
——, with Senevet & Abonnenc, (870) (Misc.
Chabeuf & Linhard, 803 (Misc.)

Chacon, R. V., with Mazza, 888 (Tryp.)
Chambers, L. A., with Plotz, Smadel & Anderson, 685 (Typh.)
Chandler, A. C., 323, 705 (Hel.)
Chandy, P. J., (317) (Lep.)
Chang, K., Tong, W. K., Li, C. H. & Chin, H. T., 851 (Hel.)
Chapman, C. J., 454 (Chl.)
Charters, A. D., 856 (Diet.)
——, with Walker, 869 (Misc.)
Chaudhuri, R. N., with Napier, 936 (Diet.)
Chávez, F., 657 (Rab.)
Chen, H. T., 254 (Hel.)
Chenoweth, B. M., Jr., with Poth & Knotts, 396 (Dys.)
Chiari, H., 444 (Typh.)
Chin, H. T., with Chang, Tong & Li, 851 (Hel.)
Chin, T. H. & Li, K. C., 847 (Hel.)
Chomet, B., 403 (Hel.)
Chopra, G. S., with Chopra, R. N. & Chopra, I. C., 87 (Misc.)
Chopra, I. C., with Chopra, R. N. & Chopra, G. S., 87 (Misc.)
Chopra, R. N., Chopra, G. S. & Chopra, I. C., 87 (Misc.)
Chorine, V., 917 (R.F.)
—— & Crougue, O., 917 (R.F.)
——, with Roubaud & Guiraud, 109 (Mal.)
Chorley, J. K., 588 (Tryp.)
Chow, C. Y., with Sweet, Feng & Hsu, 666 (Mal.)
Chow, H.-K., with Chung, 229 (Leish.)
Chowan, J. S., 172 (Vms.)
Chung, H.-L., 228 (Leish.)
—— & Chow, H-K., 229 (Leish.)
——, Wang, C. W. & Lee, C. U., 229 (Leish.)
Cifuentes, O., with Macchiavello, 593 (Typh.)
Cilento, R., 569 (B.R.)
Cimmino, V., 857 (Diet.), 892 (Typh.), 949 *bis* (Misc.)
Ciuca, *et al.*, 381 (Typh.)
Clapper, M. & Myers, G. B., 919 (R.F.)
Clark, B. M. & Goldberg, S., 605 (Pl.)
Clark, H. C., 607 (R.F.)
Clark, J. M. P., with Macpherson, 800 (Misc.)
Clarke, J. L., 748 (Mal.)
Clastrier, J., 460 (R.F.), 471 (Hel.), 741 (Mal.)
Clausell, D. T., with Pinto, 579 (Mal.)
Clavero, G. & Pérez Gallardo, F., 530 (Typh.), 641 (B.R.)
Clavero del Campo, G., & Pérez Gallardo, F., 952 (B.R.)
Clay, A. C., 779 (Dys.)
Cleland, J. B., (499) (Misc.)
Clench, W. J. & Kondo, Y., 635 (Misc.)
Cluver, E. H., 179 (Misc.)
Cochrane, E., (512) (Mal.)
——, with Newbold, (825) (Mal.)
Cochrane, R. G., 151, 785 (Lep.)
Coda, D., 518 (Mal.)
Cofiño Ubico, E. & Arguedas Klée, G., 712 (Diet.)
Coggeshall, L. T., 877 (Mal.)
Cohen, M., with Guy & Jacob, 926 (Hel.)
Cole, A. C. E., 229 (Leish.)
Cole, S. L., 242 (Dys.)
Collignon, E., 105, 430, 740 *bis* (Mal.)
Collier, H. O. J., Fulton, J. D. & Innes, J. R. M., 21 (Tryp.)
Collins, J. C., with Cameron, 144 (Am.)
Colonial Office, 632, (866) (Misc).
Colonieu, L., 590 (Leish.)
Coluzzi, A., 287 (Mal.)
Combiescu, D., Zotta, G., Manciulescu, E., Pop, A. & Tascau, J., 34 (Typh.)
Compton, A., 845 (Dys.)
Conge, M., with Nitti & Boyer, 57 (R.F.)
Cook, W. R., with Young & Kawasaki, 635 (Misc.)
Cooley, R. A., 783 (R.F.)
Cooray, G. H., 606 (Am.)
Corada Redondo, A., 603 (Y.F.)
Corbet, A. S., with Hinton, 950 (Misc.)
Corlette, C. E., 502 (Misc.)
Corradetti, A., 117 (Mal.)
Corrêa, R. R., with da Fonseca, 516 (Mal.)
—— & Ramos, A. S., 513 *bis* (Mal.)
Cossio, P., 773 (Pl.)
Cotter, E., 57 (Lep.)
Co Tui & Wright, A. M., 140 (Chl.)
Coulston, F., with Huff & Cantrell, 815 (Mal.)
Courtney, A. D., 852 (Hel.)
Coutinho, A. & Russo, E., 174 (Der.)
Coutinho, J. O., with Pessôa, 762 (Leish.)
Coutts, W. E., with Espildora, 493 (Oph.)
—— & Herrera, J. M., 144 (Am.)
Covell, G., 750, 752 (Mal.)
—— & Pritam Singh, 663 (Mal.)
Cowen, D., Wolf, A. & Paige, B. H., 950 (Misc.)
Cram, E. B., 618 (Hel.)
Crawford, J. W., with Hogan, 493 (Oph.)
Creagh, E. P. N., 522 (Bl.)
Criscuolo, E., with Tey & Marhuenda, 333 (Misc.)
Crougue, O., with Chorine, 917 (R.F.)
Crowell, R. L., with Hinman & Hurlbut, 17 (Mal.)
Cruichshank, R., 540 (Dys.)
Cruveilhier, L. & Viala, C., 203 (Rab.)
Culbertson, J. T., 75, 77, 794 (Hel.), 873 (B.R.)
—— & Rose, H. M., 471 (Hel.)
Cupi, N., 920 (Y. & S.)
—— & Cattapan, A., 124 (Leish.)
Cust, A. D., 585 (Bl.)

D.

Dairley, N. H., 540 (Dys.)
Dampf, A., 255 (Hel.)
Dancey, T. E., with Stern, (935) (Hel.)
Dang, M. L., 204 (Rab.)
Dantas, M., 921 (Lep.)
Dao Van Ty, with Brumpt, (359) (Mal.)
Daggert, W. I., 565 (Misc.)
Dartevelle, E., with Schwetz, 249 (Hel.)
Das Gupta, B. K., 936 (Diet.)
Das Gupta, B. M., 244 (R.F.)
Dastidar, S. K. G., 723 (Misc.)
Davey, T. H., with Walker, 432 (Mal.)
David & Pape, 19 (Tryp.)
David, W. A. L., 16, 293 (Mal.)
Davidson, J., (267) (Misc.)
Davis, D. J., 890 (Tryp.)
——, McGregor, T. & DeShazo, T., 590 (Tryp.)
Davis, G. B., 251 (Hel.)

Davis, G. E., 244, 782, 783 (R.F.), 533, 903 (Fev.)
Davis, M. I. J., with Franks, (821) (Mal.)
Davison, A. R., with Grasset, 611 (Lep.)
De, M. N., 947 (Misc.)
De, S. S., 861 (Vms.)
Deane, L. M., with Causey & Deane, M. P., 580 (Mal.)
——, with —— & Penido, 815 (Mal.)
Deane, M. P. & Causey, O. R., 581 (Mal.)
——, with —— & Deane, L. M., 580 (Mal.)
Dearborn, E. H., Kelsey, F. E., Oldham, F. K. & Geiling, E. M. K., 823 (Mal.)
DeBakey, L., with Faust, 664 (Mal.)
DeBakey, M., with Ochsner, 915 (Am.)
DeBettencourt, A. (662) (Rab.)
Degotte, J., 397 (Lep.)
—— & Zanetti, V., 495 (Misc.)
De Groat, A., 820 (Mal.)
DeKleine, W., 421 (Diet.)
Delgado Bedoya, G., 770, 907 (Bart.)
de Meillon, B., 110, 111 (Mal.), (180), (567), (636) (Misc.)
—— & Gillespie, J. C., 554 (Hel.)
Demina, N., 305 *bis* (Fev.)
—— & Levitanskaja, P. B., 305 (Fev.)
Denecke, K., 149 (Lep.), 179 (Misc.)
Deneke, T., (140) (Chl.)
Denison, N., 681 (Tryp.)
Dennig, 30 (Typh.)
Derbeneva-Ukhova, V. P., 499, 500 (Misc.)
Derrick, E. H., 918 (R.F.)
——, Smith, D. J. W. & Brown, H. E., 302 (Fev.)
DeSanctis, A. G. & Di Sant'Agnese, P. A., 802 (Misc.)
de Saram, G. S. W. & Townsend, R. F., 820 (Mal.)
Deschiens, R., 455 (Am.)
DeShazo, T., with Davis & McGregor, 590 (Tryp.)
de Silva, S., 239 (Fev.)
Detinova, T. S., 565 (Misc.)
Devignat, R., 390 (Pl.)
—— & Schoetter, M., 391 (Pl.)
Devine, J. & Fulton, J. D., 440 (Mal.)
Dey, N. C. & Maplestone, P. A., 175 (Der.)
Dharmendra, 548 (Lep.)
——, Lowe, J. & Mukherji, N., 316, 317 (Lep.)
—— & Mukherji, N., 701 (Lep.)
Diaz, M. P., 459 (Am.)
Diaz Rivera, R. S., Suàrez, R. M. & Hernández Morales, F., (267) (Misc.)
Diaz Rubio, M., 79, 80 (Diet.)
—— & Lara Roldán, L., 79 (Diet.)
Dick, J. C., 307 (Dys.)
Dickey, E. S., (935) (Hel.)
Ding, E., 597 (Fev.)
Dinnik, J. A. & Zvereva N. S., (66) (Hel.)
Di Sant'Agnese, P. A., with DeSanctis, 802 (Misc.)
Dixon, H. B. F., (248) (Lep.)
Djaparidze, P. S., 431 (Mal.)
Dobell, C., 52 (Am.)
Dochat, G. R., with Broders, Herrell & Vaughn, 798 (Der.)
Dolman, C. E., with Ranta, 540 (Chl.)
Dolmatova, A. V., 566 (Misc.)
Donald, C. & Barker, P. B., 30 (Typh.)
Donatien, A., with Parrot & Plantureux 527 (Leish.)
Donovan, A., with Hargett & Burruss, 692 (Y.F.)
Dormer, B. A., 398 (Hel.)
Dos Santos, I. A., with Lima, 510 (Mal.)
Doucet, 332 (Misc.)
Doudoroff, M., 775 (Pl.)
Douglas, J. R. & Wheeler, C. M., 695 (Pl.)
——, with Evans & Wheeler, 696 (Pl.)
Drenowsky, A. K., 121 (Leish.), 669 (Mal.), 790 (Hel.)
Dressler, M., with Rossier, 700 (Am.)
Dubarry & Giraud-Costa, 338 (Misc.)
Dubois, A. & Bruynseels, G. (176) (Der.)
—— & Gavrilov, W., 150 (Lep.)
—— & Kohn, J., 19, 294 (Tryp.)
—— & Valcke, G., (789) (Lep.)
Dubovsky, P., 297 (Leish.)
Dulaney, A. D., Stratman-Thomas, W. K. & Warr, O. S., 11 (Mal.)
Dunnahoo, G. L., 908 (Y.F.)
Duran-Jorda, F., 622 (Haem.)
Duren, A., 210, 743 (Mal.)
—— & Lejeune, E., 224 (Tryp.)
Dusenbery, E. D., with Seeler & Graessle, 756 (Mal.)
——, with —— & Malanga, 879 (Mal.)
Du Toit, C. J., 489 (Der.)
Dutt, N. K., with Iyenger & Mukerji, 81 (Vms.)
Dutt, P. C., 668 (Mal.)
Dyer, R. E., 602 (Fev.)
——, with Topping, 690 (Typh.)

E.

Eboué, F., 412 (Misc.)
Eckstein, A., 516, 517 (Mal.)
Eddy, G. W., 836 (Fev.)
Edmundo Vásquez, L., 767 (Typh.)
Egidio, C., 796 (Diet.)
Egorov, P. I., 501 (Misc.)
Ehrismann, O., 716 (Misc.)
Eichholtz, F. & Erhardt, A., 61 (Hel.)
Eliot, T. S., with Knisely & Stratman-Thomas, 584 (Mal.)
Elliott, M., 931 (Hel.)
Elliott, W. G. M., 702 (Hel.)
Ellis-Jones, D. W., with Wilson, 723 (Misc.)
Elmes, B. G. T., with Yarwood, 704 (Hel.)
Elsdon-Dew, R., 632 (Misc.)
Emmel, L., Gölz, E. & Jakob, A., 665 (Mal).
——, Jakob, A. & Gölz, H., 214 (Mal.)
Emmett, J. E., (603) (Y.F.)
Engelhardt, J. C., 612 (Hel.)
English, P. B. & Grey, L. P., 940 (Der.)
Enzer, J., 73 (Hel.)
Erasmus, J. F. P., 331 (Misc.)
Erhardt, A., 60 (Hel.)
——, with Eichholtz, 61 (Hel.)
Esaki, T., 870 (Misc.)
Escalona, E., 609 (Lep.)
Eskey, C. R., 834 (Typh.)
Espíldora, C. & Coutts, W. E., 493 (Oph.)
Estrada, A. T., 493 (Oph.)
Evans, F. C., Wheeler, C. M. & Douglas, J. R., 696 (Pl.)

Evans, R. W., with Smith, 384 (Typh.), 523 (Bl.)
Evans, T. C., with Levin, 556 (Hel.)
Evans, W., with Leedham-Green, 719 (Misc.)
Ewing, H. E. & Fox, I., 841 (Pl.)
Eyles, D. E. & Bishop, L. K., 667, 817 (Mal.)

F.

Faget, G. H., 847 (Lep.)
——, Johansen, F. A. & Ross, H., 610 (Lep.)
Fain, A., 373 (Tryp.)
—— & Bentz, 218 (Mal.)
Fairley, N. H. & Boyd, J. S. K., 540 (Dys.)
Falčao, J. de B., 889 (Tryp.)
Fales, J. H., with Sullivan & Goodhue, 337 (Misc.)
Fanta, H. & Siedek, H., 234 (Typh.)
Faust, E. C. & de Bakey, L., 664 (Mal.)
Fedder, M. L., with Melnikov & Sukhareva, 90 (Misc.)
Felicissimo, O., with Senra, 550 (Hel.)
Felix, A., 230 (Typh.), 540 (Dys.)
Feng, L. C., with Sweet, Chow & Hsu, 666 (Mal.)
Féreaud, C., 431 (Mal.)
Ferenczi, A., 217 (Mal.)
Ferguson, M. S., 924 (Hel.)
Fernandez, J. M. M. & Olmos Castro, N., 245 (Lep.)
Ferrari, J., with Sordelli, Manzullo & Riesel, 385 (Typh.)
Ferreira, C., 524 (Tryp.)
Ferreira, L. A., 630 (Der.)
Ferri, L. V., 459 (Am.)
Ferro-Luzzi, G., 77 (Diet.), 135 (Fev.), 854, 855 *bis*, 857 *bis* (Diet.)
Fidanza, E. P., 788 (Lep.)
Field, J. W., 6 (Mal.)
de Figueiredo, F. V., 261 *bis* (Sp.)
de Figueiredo, J., 946 (Misc.)
Figuerero, M., with Toranzos & Barbará, 680 (Tryp.)
Fillmore, R. S., with Gingrich, 439 (Mal.)
Findlay, G. M., 389 (Y.F.)
——, with Maegraith & Martin, 440, 584, 826 (Bl.)
——, Reid, R. D. & Maegraith, B. G., 593 (Typh.)
Fine, J. & Livny, 926 (Hel.)
Finkelstein, M. B., with Smyth, Gould, Koppa & Leeder, 780 (Dys.)
Finsterwalder, C., (777) (Pl.)
Fischbach, H.-W., 638 (Misc.)
Fischer, H., with Süpfle, 688 (Typh.)
Fischer, O., 93 (Misc.)
Fischthal, J. H., 794 (Hel.)
Fite, G. L., 787 (Lep.)
Fitzpatrick, F., with Gold, 32 (Typh.)
Floch, H., 768 (Typh.)
Fodor, O., with Hatieganu, 619 (Hel.)
Foley, H. & Parrot, L., 941 (Oph.)
Folha Medica, 824 (Mal.)
Follis, R. H., Jr., Miller, M. H., Wintrobe, M. M. & Stein, H. J., 795 (Diet.)
da Fonseca, F. & Corrêa, R. R., 516 (Mal.)
Fonseca, J. A. B., 515 (Mal.)
Ford, E., 412 (Misc.)
Forman, L. H., with Hellwig, 169 (Diet.)
Fort, M., with Schwetz & Baumann, 743 (Mal.)
——, with ——, —— & Beumer, 213, 357 (Mal.)
Fosdick, R. B., 112 (Mal.), (343) (B.R.)
Fox, H., (248) (Lep.)
Fox, I., 805 (Misc.)
——, with Ewing, 841 (Pl.)
Fox, J. P., 772 *bis* (Y.F.)
—— & Cabral, A. S., 451 (Y.F.)
——, Lennette, E. H., Manso, C. & Aguiar, J. R. S., 44 (Y.F.)
——, Manso, C., Penna, H. A. & Pará, M., 41 (Y.F.)
Foy, H., Altmann, A., Barnes, H. D. & Kondi, A., 363 (Bl.)
—— & Kondi, A., 883 (Bl.)
Franceschi, C., with Pirosky & Sampayo, 266 (Vms.)
Franklin, A. V., 205 (Rab.)
Franklin, L. B., with Turnbull, 340 (Misc.)
Franks, A. G. & Davis, M. I. J., (821) (Mal.)
—— & Taylor, H. G., 940 (Der.)
Frear, D. E. H., 807 (B.R.)
Freeman, W., with Wien & Scotcher, 683 (Leish.)
Freudenthal, E., 492 (Oph.)
Frewen, W. K., 585 (Bl.)
Frey, J. R., with Prunes, 491 (Der.)
Fróes, J. A. G., 442 (Typh.)
Frohne, W. C., 215 (Mal.)
Fuentes, C. & José Angulo, J., 630 (Der.)
Fuller, H. S. & Geiman, Q. M., 230 (Leish.)
Fulton, J. D., 683 (Leish.)
——, with Collier & Innes, 21 (Tryp.)
——, with Devine, 440 (Mal.)
—— & Yorke, W., 19 (Tryp.), 23 (Leish.), 754 (Mal.)

G.

Gaase, A., 329 *bis* (Hel.)
Gajardo-Tobar, R., 83 (Vms.)
Gallardo, F. P., 894 (Typh.)
Gallia, F., with Kubes, 208, 661 (Rab.)
Galliard, H., 71 (Hel.)
Gallut, J., 910 (Chl.)
Gandrille, with d'Allaines & Lavier, 472 (Hel.)
Ganguly, S. C., with Gupta & Kahali, 677 (Bl.)
Ganguli, S. K., with Roy, 749 (Mal.)
Garzon, R. & Molina, R. J., 125 (Leish.)
Gasic, G., 888 (Tryp.)
—— & Carvajal, V., 760 (Tryp.)
Gasperini, G. C., (215) (Mal.), 916 (R.F.)
Gaud, J., 598 (Typh.)
——, with Barnéoud, 469 (Hel.)
Gavrilov, W., with Dubois, 150 (Lep.)
Gaynon, I. E., with Weiner & Osherwitz, 493 (Oph.)
Gay Prieto, J., López de Azcona, J. M., Azúa, L., Bondeu, F. & Cárdenas, M., 94 (Misc.)
Gear, J., 591 (Typh.)
Gehr, E., 59 (Lep.)
—— & Gehr, E., 60 (Lep.)
Geiling, E. M. K., with Dearborn, Kelsey & Oldham, 823 (Mal.)

Geiman, Q. M., with Fuller, 230 (Leish.)
Gelber, A., 377 (Leish.)
Gelfand, M., 398 (Hel.)
Gellis, S., with Riordan & Rubinowitz, 859 (Diet.)
Genezzani, U., (722) (Misc.)
Georgevic, I., 291 (Mal.)
Ghosh, B. K., with Mukerji, 922 (Hel.)
——, with —— & Siddons, 670 (Mal.)
Ghosh, B. N., with Sarkar & Maitra, 265 (Vms.)
Ghosh, G., 268 (Misc.)
Ghosh, S. K., with Panja, 910 (Chl.)
Ghosh, S. M., with Roy, 413 (Misc.)
Gibbins, E. G., 304 (Y.F.)
Gibson, E. B., with Randolph, 860 (Haem.)
Gilbert, F. W., 589 (Tryp.)
Gildemeister, E. & Peter, H., 895 (Typh.)
Gilkerson, S. W., with Boyd, 221 (Mal.)
Gill, C. E., with Hayes, (36) (Typh.)
Gillespie, J. C., with de Meillon, 554 (Hel.)
Gillet, R., (609) (Lep.)
Gilman, R. L., 86 (Misc.)
Gingrich, W. D. & Fillmore, R. S., 439 (Mal.)
Ginsburg, J. M., 337 (Misc.)
Girard, G., 139, 842 (Pl.)
—— & Robic, J., 777 (Pl.)
Giraud-Costa, with Dubarry, 338 (Misc.)
Giri, K. V., with Khorana & Sarma, 80 (Diet.)
Giroud, P., 36 (Typh.)
—— & Panthier, R., 35 *bis*, 764 (Typh.).
Glaubach, N. & Guller, E. J., 181 (Misc.)
Glazko, A. J., 880 (Mal.)
Glover, J., 502 (Misc.)
Gnezdilov, V. G., 911 (Am.)
Godard, G., with Lafuente, 892 (Leish.)
Godoy, S. G., 746 (Mal.)
Goeters, W., 596 (Fev.)
Gokhale, P. V., 867 (Misc.)
Gold, H. & Fitzpatrick, F., 32 (Typh.)
Goldberg, S., with Clark, 605 (Pl.)
Golden, W. R. C., with Watt, Olason & Mladinich, 793 (Hel.)
Goldmann, C. H. & Smith, S. J., 919 (Y. & S.)
Goldsmith, G. A., 559 (Diet.)
Gölz, H., with Emmel & Jakob, 214, 665 (Mal.)
Gönnert, R., 160 (Hel.)
Gontaeva, A. A., 436 (Mal.)
González, E., with Aguilar Meza & Medrano, 292 (Mal.)
Gonzalez Guzman, I., 787 (Lep.)
Gonzalez Ochoa, A., 489 (Der.)
Good, N. E., with Jellison, (180) (Misc.)
Goodhue, L. D., with Sullivan & Fales, 337 (Misc.)
Goodwin, M. H., Jr., 581 (Mal.)
Goodwin, T. W., 684 (Leish.)
Gordon, E., 311 (Am.)
Gordon, J. & Gordon, M., 392 (Chl.)
Gordon, S., with Young, 73 (Hel.)
Goritzkaya, V. V., 433 (Mal.)
Gotor, P., 65 (Hel.)
Gould, S. E., 619 (Hel.)
——, with Smyth, Finkelstein, Koppa & Leeder, 780 (Dys.)
Gracian, M., 297 (Typh.)
Graessle, O., with Seeler & Dusenbery, 756 (Mal.)
Grant, A. & Barwell, C., 498 (Misc.)
Grant, L. I. H., 932 (Hel.)
Grasset, E. & Davison, A. R., 611 (Lep.)
Gregg, A. L., 641 (B.R.)
Gregory, M. K., 858 (Diet.)
Grey, L. P., with English, 940 (Der.)
Grindley, D. N., with Henry, 122 (Leish.)
Groover, M. E., Jr., with Sheldon, 321 (Hel.)
Gros, G., with Senevet, Moutier & Bourgarel, 805 (Misc.)
Grubmüller, J., 899 (Typh.)
Grysez, V., 198 (Rab.)
Guardascione, V., 218 (Mal.)
Guigon, G., with Méchin, 527 (Leish.)
Guilhaumou, F., with Ceccaldi, 56 (R.F.)
Guindy, 139 (Chl.), 220 (Mal.)
Guiraud, P., with Roubaud & Chorine, 109 (Mal.)
Guller, E. J., with Glaubach, 181 (Misc.)
Günther, L., with Walther, 51 (Dys.)
Guntscheff, N., 93 (Misc.)
Gupta, J. C., Kahali, B. & Ganguly, S. C., 677 (Bl.)
Gupta, S. K., with Napier, 539 (Chl.)
Guy, W. H., Cohen, M. & Jacob, F. M., 926 (Hel.)
György, P., with Caldwell, 891 (Tryp.)

H.

Hackett, C. J., 631 (Misc.)
Haddow, A. J., 816 (Mal.)
Hafez, M., 91, (93) (Misc.)
Hakansson, E. G., 268 (Misc.)
—— with Sapero & Louttit, 53 (Am.)
Hallmann, 694 (Fev.)
Halter, B. L. & Kuzell, W. C., 862 (Vms.)
Hamilton, A. H. 750 (Mal.)
Hamilton, J. B., 461 (R.F.)
Hamilton, J. L., with Burt & Lane, 923 (Hel.)
Hampil, B. & Roberts, G. H., 200 (Rab.)
Hanes, F. M., 170 (Sp.)
Hansen-Pruss, O. C., 860 (Diet.)
Hardwick, S. W., 796 (Diet.)
Hargett, M. V., Burruss, H. W. & Donovan, A., 692 (Y.F.)
Harington, C., with Simson & Barnetson, 630 (Der.)
Harmsen, with Linneweh, 935 (Hel.)
Harmsen, H. & Hauer, A., 669 (Mal.)
Harrell, W. B. & Moseley, V., 93 (Misc.)
Harries, E. H. R., with Van den Ende, Stuart-Harris & Steigman, 449 (Typh.)
Harris, C. E. S., with Stanley-Jones, 81 (Vms.)
Harris, W. H., Jr., 304 (Y.F.)
Harris, W. V., 39 (Y.F.)
Haseman, L., with Pearman, (724) (Misc.)
Hasselmann, C. M., 12 (Mal.)
Hatheway, A. E., with Manwell, 883 (Mal.)
Hatieganu, J. & Fodor, O., 619 (Hel.)
Hauer, A., 913 (Am.)
——, with Harmsen, 669 (Mal.)
Hawking, F., 616 (Hel.) 823 (Mal.)
Hayes, J. E. & Gill, C. E., (36) (Typh.)
Haythornthwaite, H. M., 702 (Lep.)
Headlee, W. H., 148 (Am.), 710 (Hel.)
—— & Cable, R. M., 148 (Am.)

Hecht O., 605 (Pl.)
Hedges, H. S. & Humphries, M. K., Jr., (340) (Misc.)
Heilig, R., 68 (Hel.), 711 (Diet.)
—— & Visweswar, 360 (Mal.), 400 (Hel.), 406 (Haem.)
Heiss, F., 94 (Misc.)
Hellwig, C. A. & Forman, L. H., 169 (Diet.)
Hemphill, J. E. & Noojin, R. O., 562 (Der.)
Henderson, J. M., 718 (Misc.)
Hennessey, R. S. F., 240 (Pl.)
Henrard, C., with van Hoof & Peel, 367, 368 (Tryp.)
Henry, A. J. & Grindley, D. N., 122 (Leish.)
Henschel, E. J., with Silverberg, 377 (Leish.)
Henschke, U., 568 (Misc.)
Herbut, P. A., with Reimann & Price, 853 (Hel.)
Hermann, H. B. & Berman, L. S., 457 (Am.)
Hernández Morales, F., with Diaz Rivera & Suárez, (267) (Misc.)
Herrell, W. E., with Broders, Dochat & Vaughn, 798 (Der.)
Herrera, J. M., with Coutts, 144 (Am.)
Herrera, J. R., with Bustamante & Kumm, 453 (Y.F.)
——, with Kumm & Bustamante, (878) (Mal.)
Herzog, E., 199 (Rab.)
Hewitt, R., 222 (Mal.)
——, with Hurlbut, 583 (Mal.)
Hewitt, R. I., Richardsou, A. P. & Seager, L. D., 439 (Mal.)
Hill, A. W. & Andrews, J., 324 (Hel.)
Hill, C. M., 293 (Mal.)
Hill, R. B., Cambournac, J. C. & Simões, M. P., 818 (Mal.)
——, with Carr, 745 (Mal.)
Hill, T. R., 634 (Misc.)
Hinman, E. H., Crowell, R. L. & Hurlbut, H. S., 17 (Mal.)
Hinton, H. E. & Corbet, A. S., 950 (Misc.)
Hirsh, J., 872 (Misc.)
Hoff, F. & von Brunn, E., 299 (Typh.)
Hoffman, W. A., with Krakower & Axtmayer, 923 (Hel.)
Hogan, M. J. & Crawford, J. W., 493 (Oph.)
Holcomb, R. C., (176) (Der.)
Hopkins, G. H. E., 671, 672 (Mal.)
Horrenberger, R., with Béguet, 657 (Rab.)
Hosford, G. N., Stewârt, M. A. & Sugarman, E. I., 257 (Hel.)
Hou, H. C., 404 *bis* (Diet.)
Howard, R. S., Jr. & Andrews, J., 16 (Mal.)
——, with Legwen, 674 (Mal.)
——, with —— & Turner, 673 (Mal.)
Howard, S., 157 (Hel.)
Howe, C., 137, 905 (Bart.)
Howie, J. W., with Smith, 303 (Y.F.)
Hsiung, J. C. & Kimm, H. T., (267) (Misc.).
Hsu, S. C., with Sweet, Feng & Chow, 666 (Mal.)
Hu, S. M. K., 254 (Hel.)
Huff, C. G., Coulston, F. & Cantrell, W., 815 (Mal.)
Hughes, T. P., 771 (Y.F.)
Hughes, W. & Murgatroyd, F., 754 (Mal.)
Hühne, W., 292 (Mal.)
Humphries, M. K., Jr., with Hedges, (340) (Misc.)
Hurlbut, H. S., 748 (Mal.)
—— & Hewitt, R., 583 (Mal.)
——, with Hinman & Crowell, 17 (Mal.)

I.

Ignacio Chala, J., 248 (Lep.)
India, 784 (Lep.)
Indian Journal of Medical Research, 227 (Leish.)
Indian Medical Gazette, (23) (Leish.), 360 (Hel.)
Ingelfinger, F. J., 712 (Sp.)
—— & Moss, R. E., 937 (Sp.)
Innes, J. R. M., with Collier & Fulton, 21 (Tryp.)
Irdem, E., 816 (Mal.)
Iriarte, D. R., 25 (Leish.), 375, (891) (Tryp.), 546 (Y. & S.).
Iyengar, M. O. T., 663 (Mal.)
Iyenger, N. K., Dutt, N. K. & Mukerji, B., 81 (Vms.)
Izac, R., 942 (Oph.)
——, with Siegfried, 105, 738 (Mal.)

J.

Jack, R. W., 524 (Tryp.)
Jackson, W. P., 563 (Misc.)
Jacob, F. M., with Guy & Cohen, 926 (Hel.)
Jacob, V. P., with Russell, 105 (Mal.)
Jaffé, W., 612 (Hel.)
Jagujinskaia, L. W., 179 (Misc.)
Jakob, A., with Emmel & Golz, 214, 665 (Mal.)
Jamieson, R. C. & McCrea, A., 175 (Der.)
Janssens, P. G., with Vincke, 391 (Pl.)
Javett, S. N. & Sacks, S., 216 (Mal.)
Jellison, W. L. & Good, N. E., (180) (Misc.)
Jiménez-Martínez, P., 447 (Fev.)
Jiménez-Diaz, C., Roda, E., Ortiz de Landázuri, E., Marina, C. & Lorente, L., 87 (Misc.)
—— & Vivanco, F., 87 (Misc.)
Jirovec, O., with Kučera, 700 (Am.)
Johansen, F. A., with Faget & Ross, 610 (Lep.)
Johnson, A. S., 947 (Misc.)
Johnson, D. W., 918 (R.F.)
Johnson, H. A., with Robertson, LePrince & Parker, 15 (Mal.)
Johnson, H. N., 207, 660 (Rab.)
——, with Leach, 655 (Rab.)
Jolly, R. H. H., 619 (Hel.)
Jones, F., with Rees, Bozicevich & Reardon, 456 (Am.)
Jones, M. F., with Brady & Newton, 782 (Am.)
Jordan, K., (240) (Pl.)
José Angulo, with Fuentes, 630 (Der.)
Joukovsky, T., with Barlovatz, 37 (Fev.)
Journal of the American Medical Association, 40 (Y.F.), 582 (Mal.)
Joynt, E. P. H., 865 (Misc.)
Julianelle, L. A., 941 *bis* (Oph.)
Junior, M. S., 50 (Pl.)
Junqueira, M., with Versiani, 890 (Tryp.)

K.

Kahali, B., with Gupta & Ganguly, 677 (Bl.)
Kamalova, A. G., with Podyapolskaya, 474 (Hel.)
Kammer, V. M., 607 (R.F.)
Kasimow, G. B., 326 (Hel.)
Kauer, G. L., Jr., Bird, R. M. & Reznikoff, P., 560 (Vms.)
Kaufman, W. H. & Smith, D. C., 483 (Sp.)
Kawasaki, I. A., with Young & Cook, 635 (Misc.)
Kellaway, C. H., 263 (Vms.)
Kellner, F., 948 (Misc.)
Kelsey, F. E., with Dearborn, Oldham & Geiling, 823 (Mal.)
Kerger, H., 136 (Fev.)
Kermack, W. O., 179 (Misc.)
Kern, R., with Youmans, Patton & Robinson, 620 (Diet.)
Kessler, A., 628 (Der.)
Key, J. A. & Large, A. M., 177 (Der.)
Khorana, M. L., Sarma, M. L. & Giri, K. V., 80 (Diet.)
Kikuth, W., 284 (Mal.)
—— & Mudrow, L., 677 (Mal.)
Kilham, L., 217 (Mal.)
Kimm, H. T., with Hsiung, (267) (Misc.)
King, H. & Strangeways, W. I., 20 (Tryp.)
Kirk, R., 377 (Leish.)
—— & Sati, M. H., 684 (Leish.)
Kirsner, J. B., with Rodaniche, 455 (Am.)
Kitchen, S. F., with Boyd, 818 (Mal.)
—— & Putnam, P., 109 (Mal.)
Kligler, I. J. & Bernkopf, H., 651 (Rab.)
Knipe, F. W. & Russell, P. F., 115, 673 (Mal.)
——, with —— & Rao, 14, 114, 753 (Mal.)
—— & Sitapathy, N. R., 116 (Mal.)
Knipling, E. F., with Brody, 636 (Misc.)
Knisely, M. H. & Bloch, E. H., 584 (Mal.)
——, Stratman-Thomas, W. K. & Eliot, T. S., 584 (Mal.)
Knotts, F. L. with Poth & Chenoweth, 396 (Dys.)
Kohls, G. M., with Parker & Steinhaus, 835 (Fev.)
Kohn, I., with Dubois, 19, 294 (Tryp.)
Koivastik, T., with Peterson, 624 (Vms.)
Komp, W. H. W., 334 (Misc.), 808 (B.R.), 818 (Mal.)
Kondi, A. with Foy, 883 (Bl.)
——, with Foy, Altmann & Barnes, 363 (Bl.)
Kondo, Y., with Clench, 635 (Misc.)
Kopp, I., 10 (Mal.)
—— & Solomon, H. C., 582 (Mal.)
Koppa, T. M., with Smyth, Finkelstein, Gould & Leeder, 780 (Dys.)
Koppisch, E., 703 (Hel.)
Kowalzig, 120 (Leish.)
Krakower, C., Hoffman, W. A. & Axtmayer, J. H., 923 (Hel.)
——, Morales-Otero, P. & Axtmayer, J. H., 789 (Lep.)
Krasikova, V. I., 434 (Mal.)
Kriukova, A. P., with Latyshev, 24, 296 (Leish.)
Kruse, H. D., 169 (Diet.)
Kubes, V. & Gallia, F., 208, 661 (Rab.)
Kučera, K. & Jirovec, O., 700 (Am.)
Kuhlmann, F., with Abicht, 937 (Sp.)
Kuitunen-Ekbaum, E., 710 (Hel.)
—— & Morgan, E. M., 328 (Hel.)
Kulkarni, S. S., with Row, 378 (Leish.)
Kumm, H. W., 359 (Mal.)
——, Bustamante & Herrera (878) (Mal.)
——, with Bustamante & Herrera, 453 (Y.F.)
—— & Zúniga, H., 108 (Mal.)
Kunert, H. & Buch, E., 130 (Typh.)
Kuptzova, A. D., with Blakhov, 435 (Mal.)
Kuzell, W. C., with Halter, 862 (Vms.)

L.

Lack, A. R., Jr., 676 (Mal.)
Laemmert, H. W., Jr., 694 (Y.F.)
Lafuente, M. & Godard, G., 892 (Leish.)
Lahiri, M. N., 783 (R.F.)
Lampert, H., 595, 831 (Typh.)
Landsberg, E., with Morrison & Samwick, 407 (Haem.)
Lane, J. M., with Burt & Hamilton, 923 (Hel.)
Langley, G. F., (614) (Hel.)
Lapuishev, D. A., 871 (Misc.)
Lara, M. B., 464 (Lep.)
Lara Roldán, L., with Diaz Rubio, 79 (Diet.)
Large, A. M., with Key, 177 (Der.)
Larsh, J. E., Jr., 322, 851 (Hel.)
Lasnier, E. P. & Cassinelli, J. F., 614 (Hel.)
Lassman, P., with Rodhain, 119 (Mal.)
Latyshev, N. I. & Kriukova, A. P., 24, 296 (Leish.)
Laurentius, P., 445 (Typh.)
Lavier, G., with d'Allaines & Gandrille, 472 (Hel.)
Lavrenko, E. M., 510 (Mal.)
Lawler, N. A., with Cameron, 458 (Am.)
Lawson, T. L., 224 (Tryp.)
Leach, C. N. & Johnson, H. N., 655 (Rab.)
League of Nations, 126 (Typh.)
Lee, C. U., with Chung & Wang, 229 (Leish.)
Leeder, F. S., with Smyth, Finkelstein, Gould & Koppa, 780 (Dys.)
Leedham-Green, J. C. & Evans, W., 719 (Misc.)
Legwen, W. A. & Howard, R. S., Jr., 674 (Mal.)
—— & Lenert, L. G., 673 (Mal.)
Leider, A. G., with Schaeffer, 199 (Rab.)
Lejeune, E., with Duren, 224 (Tryp.)
Lenert, L. G., with Legwen, 673 (Mal.)
Lennette, E. H., with Fox, Manso & Aguiar, 44 (Y.F.)
Lent, H., (526) (Tryp.)
Leon, A. P., 382 (Typh.)
Leon, E. R., with Pardo-Castello & Trespalacios, 174 (Der.)
LePrince, J. A., with Robertson, Johnson & Parker, 15 (Mal.)
Leprosy in India, (546), 701 (Lep.)
Lerner, J., with Alessandri & Palazuelos, 168 (Diet.)
Levaditi, C., 206 *bis* (Rab.)
Lever, R. J. A. W., 816 (Mal.)
Levin, A. J. & Evans, T. C., 556 (Hel.)
Levin, M. B., 794 (Hel.)

Levitanskaja, P. B., with Demina, 305 (Fev.)
Lewicki, E., 531 (Typh.)
Lewis, D. J., 288, 665 (Mal.), 334 (Misc.), 602 (Y.F.)
Lewis, E. A., 293, 294, 678 (Tryp.)
——, Wiley, A. J. & Macaulay, J. W., 887 (Tryp.)
Li, C. C., with Pollitzer, 776 (Pl.)
Li, C. H., with Chang, Tong & Chin, 851 (Hel.)
Li, K. C., with Chin, 847 (Hel.)
Lilga, H. V., with Blum, 552 (Hel.)
Lima, F. de O. & Dos Santos, I. A., 510 (Mal.)
Limboś, P., with Radna, 316 (R. F.)
Lincicome, D. R., 456 (Am.)
Lindsay, D. K., 878 (Mal.)
Linhard, with Chabeuf, 803 (Misc.)
Linhares, H., (922) *bis* (Lep.)
Linneweh, F. & Harmsen, 935 (Hel.)
Litchfield, J. T., Jr., with Marshall & White, 223 (Mal.)
Livny, with Fine, 926 (Hel.)
Lockhart, J., with McDermott, Webster, Baker & Tompsett, 679 (Tryp.)
Löffler, W. & Mooser, H., 28 (Typh.)
Loos, W., 532 (Typh.)
López de Azcona, J. M., with Gay Prieto, Azúa, Bondeu & Cárdenas, 94 (Misc.)
Lopez-Neyra, C. R., 614 (Hel.)
Lopez Portillo, S., 315 (R.F.)
——, S., with Beltran, 459 (Am.)
Lorente, L., with Jiménez-Diaz, Roda, Ortiz de Landázuri & Marina, 87 (Misc.)
Lourie, E. M., 370 (Tryp.)
Loutit, J. F., 523 (Bl.)
Louttit, C. M., with Sapero & Hakansson, 53 (Am.)
Lowe, J., (51) (Pl.), 317 (Lep.)
——, with Dharmendra & Mukherji, 316, 317 (Lep.)
Lubinsky, G. A., 613 (Hel.)
Lucas, T. L., 181 (Misc.)
Lund, H. O., 581 (Mal.)
Luttermoser, G. W., 849 (Hel.)
Lydtin, 384 (Typh.)
Lyon, G. M., 142, 543 (Dys.)

M.

Macaulay, J. W., 885 (Tryp.)
——, with Lewis & Wiley, 887 (Tryp.)
McCarthy, D. F., 762 (Typh.)
McCartney, J. L., 802 (Misc.)
Macchiavello, A., 46 *bis*, 47 *bis*, 48, 49, 909 (Pl.)
—— & Cifuentes, O., 593 (Typh.)
—— & Paracampos, H., 48, 49 *bis*, 50 (Pl.)
McCoy, G. W., 465, 609 (Lep.)
McCoy, O. R., 258 (Hel.)
McCrea, A., with Jamieson, 175 (Der.)
McCulloch, R. N., with Walker, Meyers & Woodhill, (604) (Fev.)
McDermott, W., Webster, B., Baker, R., Lockhart, J. & Tompsett, R., 679 (Tryp.)
McDonald P. R., with Machella, 559 (Diet.)
Macedo, E., with Viana Martins, 761 (Tryp.)
McGovran, E. R. & Sullivan, W. N., 338 (Misc.)
McGregor, T., with Davis & DeShazo, 590 (Tryp.)
Machella, T. E. & McDonald, P. R., 559 (Diet.)
Mackay, A. G., 155 (Lep.)
McKendon, S. B., with Young & Smart, 881 (Mal.)
Mackenzie, M. D., (130) (Typh.)
MacLean, K. S., 719 (Misc.)
McLendon, S. B., 756 (Mal.)
MacNalty, A., (579) (Mal.)
Macpherson, I. & Clark, J. M. P., 800 (Misc.)
Madras, 494 (Oph.)
Maegraith, B., Findlay, G. M. & Martin, N. H., 440, 584 (Bl.)
——, with Findlay & Reid, 593 (Typh.)
——, Martin, N. H. & Findlay, G. M., 826 (Bl.)
de Magalhães, O., 82 (Vms.)
—— & Rocha, A., (136), 300 (Typh.)
Magath, T. B., 156 (Hel.)
Maher, H. C., with Watson & Rice, 665 (Mal.)
Maitra, M. N., with Sanyal, 492 (Oph.)
Maitra, S. R., with Sarkar & Ghosh, 265 (Vms.)
Malanga, C., with Seeler & Dusenbery, 879 (Mal.)
Malbrant, R., 588 (Tryp.)
Malik, K. S., with Panja & Paul, 140 (Chl.)
Maly, G., 236 (Typh.)
Manalang, J., 465 (Lep.)
Manciulescu, E., with Combiescu, Zotta, Pop & Tascau, 34 (Typh.)
Manine, A., (566) (Misc.)
Manso, C., with Fox, Lennette & Aguiar, 44 (Y.F.)
——, with ——, Penna & Pará, 41 (Y.F.)
Manson-Bahr, P. H., (9) (Mal.), 415, 540 (B.R.)
Manwell, R. D., 756 (Mal.)
—— & Hatheway, A. E., 883 (Mal.)
Manzelli, M. A., 335 (Misc.)
Manzullo, A., with Sordelli, Riesel & Ferrari, 385 (Typh.)
Maplestone, P. A., (923) (Hel.)
——, with Dey, 175 (Der.)
——, with Mukerji, 707, 925 (Hel.)
Marble, A., Skoog, A. P. & Bucholz, D. J., 258 (Hel.)
Marchionini, A., 125 (Leish.), 339 (Misc.)
Marhuenda, P., with Tey & Criscuolo, 333 (Misc.)
Marie, P. L., 903 (Fev.)
Marill, F-G., with Alcay, L. & Musso, J.-C., 467 (Hel.)
Marina, C., with Jiménez-Diaz, Roda, Ortiz de Landázuri & Lorente, 87 (Misc.)
Markaryan, A., with Matevossian & Sarkissian, 310 (Am.)
Markovich, N. Y., 519 (Mal.)
Marshall, E. K., Jr., 223 (Mal.)
——, Litchfield, J. T., Jr. & White, H. J., 223 (Mal.)
Marshall, J. F., 870 (Misc.)
Martin, N. H., with Maegraith & Findlay, 440, 584, 826 (Bl.)
Martin, R., 744 (Mal.)
Martínez Báez, M., 922 (Lep.)
——, with Millan Gutierrez & Salazar Mallen, 482 (Diet.)
Martinic, A., (853) (Hel.)

Martins, A. V., 939 (Vms.)
Martín Sánchez, A., 848 (Hel.)
Mason, H. C., 197 (Rab.)
Mason, H. L., with Williams, Power & Wilder, 558 (Diet.)
Masse, G., 148 (Y. & S.)
Matevosian, C. M., (110) (Mal.)
Matevossian, S., Sarkissian, M. & Markaryan, A., 310 (Am.)
Mathevossian, E. M., with Skrjabin, (400) *ter* (Hel.)
Mauss, E. A. & Otto, G. F., 328 (Hel.)
Mazza, S., 525, 761 (Tryp.)
——, Basso, G. & Basso, R., 22, 226 (Tryp.)
—— & Chacon, R. V., 888 (Tryp.)
Mazzotti, L., 253, 476 (Hel.) 314, 545 (R.F.), 891 (Tryp.)
—— & Osorio, M. T., 617, (851) (Hel.)
—— & Varela, G., 301 (Typh.)
Mears, A. R. R., 405 (Diet.), 865 (Misc.)
Mears, G., 865 (Misc.)
Méchin, R. & Guigon, G., 527 (Leish.)
Meeser, C. C. V., 475 (Hel.)
Medical Research Council, 699 (Am.), 867 (Misc.)
Mednikian, G. A., (173) (Vms.)
Medrano, A. R., with Aguilar Meza & González, 292 (Mal.)
Megaw, J. W. D., 27 (Typh.)
Melcher, L. R. & Campbell, D. H., 328 (Hel.)
Meleney, H. E., (85) (Misc.)
Melnikov, N. N., Sukhareva, N. D. & Fedder, M. L., 90 (Misc.)
de Mendonça, J. M., 171 (Haem.)
Mendoza, L., 136 *bis*, 767 (Typh.), 267 (Misc.)
Meng, C. H. & Winfield, G. F., 871 (Misc.)
Menk, W., (14) (Mal.)
Menon, K. P., Ayyar, P. V. S. & Shortt, H. E., 220 (Mal.)
Menon, P. B., with Russell, 362 (Mal.)
Mera, B., 904 (Bart.)
de Mesquita, B., 744 (Mal.)
Messerlin, with Sicault, (284) (Mal.)
Messerlin, A., 742, 753 (Mal.)
Messerschmidt, T., with Schütz, 132 (Typh.)
Mevzos, M. P., 824 (Mal.)
Meyer, R., 689 (Typh.)
Meyers, E., with Walker, Woodhill & McCulloch, (604) (Fev.)
Mihaljevič & Radičev, 446 (Typh.)
Millan Gutierrez, J., Salazar Mallen, M. & Martinez Baez, M., 482 (Diet.)
Miller, M. H., with Follis, Jr., Wintrobe & Stein, 795 (Diet.)
Mills, C. A. & Schmidt, L. H., 567 (Misc.)
Minett, J. S., 313 (R.F.)
Minning, W., 63 (Hel.)
Mirzoian, N. A., 295 (Leish.)
Missiroli, A., 221 (Mal.)
Mitton, R. E. R. & Beattie, M. K., 942 (Oph.)
Mixter, W. J., with Shattuck, 953 (B.R.)
Mladinich, G., with Watt, Golden & Olason, 793 (Hel.)
Mogilnizky, B. N. & Brumstein, M. S., 141 (Dys.)
Mohammed, A. H., 82 (486) (Vms.),
Mohan, B. N., with Russell, (17), 362 (Mal.)
——, with —— & Mulligan, 117 (Mal.)
Mohr, J. L., with Mumford, 716 (Misc.)
Mohr, W., 10, 13 (Mal.)
Molina, R. J., with Garzon, 125 (Leish.)
Moll, A. A., 625 (Vms.)
—— & O'Leary, S., 46, 239, 389 (Pl.)
Montel, R., 722 (Misc.), 789 (Lep.), 846 (Y. & S.).
Moore, D. F., 480 (Diet.)
Mooser, H., with Löffler, 28 (Typh.)
Moragues, V., 714 (Haem.)
Morales-Otero, P., with Krakower & Axtmayer, 789 (Lep.)
——, with Pomales-Lebrón, 528 (Typh.)
Morgan, E. M., with Kuitunen-Ekbaum, 328 (Hel.)
Morrison, M., Samwick, A. A. & Landsberg, E., 407 (Haem.)
Moseley, V., with Harrell, 93 (Misc.)
Moss, E. S., with Schenken, 555 (Hel.)
Moss, R. E., with Ingelfinger, 937 (Sp.)
Most, H., 930 (Hel.)
Moustardier, G., 56 (R.F.), 594 (Typh.)
Moutier, P., with Senevet, Alcay, Gros & Bourgarel, 805 (Misc.)
Mudrow, L., 116 (Mal.)
——, with Kikuth, 677 (Mal.)
Muir, E., 462, 463 *quin.* (Lep.)
Mukerjee, S., with Ray & Roy, 118 (Mal.)
Mukerji, A. K. & Ghosh, B. K., 922 (Hel.)
—— & Maplestone, P. A., 707, 925 (Hel.)
Mukerji, B., Ghosh, B. K. & Siddons, L. B., 670 (Mal.)
——, with Iyenger & Dutt, 81 (Vms.)
Mukherji, N., with Dharmendra, 701 (Lep.)
——, with —— & Lowe, 316, 317 (Lep.)
Mulligan, H. W., with Russell & Mohan, 117 (Mal.)
Mumford, E. P., (9), 107 (Mal.)
—— & Mohr, J. L., 716 (Misc.)
Mumme, C. & Sundermann, A., 328 (Hel.)
Muñoz Rivas, G., 151 (Lep.), 486 (Fev.)
Muraz, G., 524 (Tryp.), 948 (Misc.)
Murgatroyd, F., 757 (Bl.)
——, with Hughes, 754 (Mal.)
Murray, J. E. & Shute, P. G., 9, 821 (Mal.)
Musso, J.-C., with Alcay & Marill, 466 (Hel.)
Myers, G. B., with Clapper, 919 (R.F.)

N.

Nagel, A., (853) (Hel.)
Naidenova, G. A. & Talysin, F. F., (173) (Vms.)
Naidu, V. R., Rao, A. V. & Rajagopal, M. D., 290 (Mal.)
Napier, L. E. & Chaudhuri, R. N., 936 (Diet.)
—— & Gupta, S. K., 539 (Chl.)
——, Sen Gupta, P. C. & Sen, G. N., 121 (Leish.)
Nayar, S., 262 (Haem.)
Nehaul, B. B. G., 442 (Leish.)
Nelson, A. J. M., 884 (Bl.)
Neumann, M. A., (850) (Hel.)
Newbold, C. E. & Cochrane, E., (825) (Mal.)
Newton, W. L., with Brady & Jones, 782 (Am.)
Nicholas, L., 872 (Misc.)
Nichols, J. B., 579 (Mal.)
Nickel, H. S., 70 (Hel.)
Nicol, R., (168) (Diet.)

Nicolay, F., 104 (Mal.)
Niño, F. L., 145 (Am.), 402 (Hel.), 715 (Der.)
—— with Brea & Taiana, 698 (Am.)
Nitti, F., Boyer, F. & Conge, M., 57 (R.F.)
Niyogi, A. K., 750, 755 (Mal.)
—— & Ray, J. C., 762 (Leish.)
—— & Roy, A. N., 755 (Mal.)
Noojin, R. O. & Callaway, J. L., 797 (Der.)
——, with Hemphill, 562 (Der.)

O.

Oberhoff, K., 494 (Oph.)
O'Brien, C. S., 943 (Oph.)
Ochsner, A. & DeBakey, M., 915 (Am.)
Officer-Brown, C. J., (795) (Hel.)
Ogden, M. A., 623 (Haem.)
Olason, F., with Watt, Golden & Mladinich, 793 (Hel.)
Oldham, F. K., with Dearborn, Kelsey & Geiling, 823 (Mal.)
O'Leary, S., with Moll, 46, 239, 389 (Pl.)
Olmos Castro, N., with Fernandez, 245 (Lep.)
Olpp, G., (108) (Mal.)
Ormiston, G., Taylor, J. & Wilson, G. S., 54 (Am.)
Ortega, F., 431 (Mal.)
Ortiz Armengol, F., 202 (Rab.)
Ortiz de Landázuri, with Jiménez-Diaz, Roda, Marina & Lorente, 87 (Misc.)
Osherwitz, with Weiner & Gaynon, 493 (Oph.)
Osorio, M. T., with Mazzotti, 617, (851) (Hel.)
Oswald, E., with Sadusk, 843 (Chl.)
Otálora, B., 759 (Tryp.)
Ott, A., 781 (Am.)
Otto, G. F., with Mauss, 328 (Hel.)
Otto, R. & Bickhardt, R., 129 (Typh.)

P.

Packchanian, A., 526 (Tryp.)
Page, R. C., with Bercovitz & de Beer, 929 (Hel.)
Paige, B. H., with Cowen & Wolf, 950 (Misc.)
Palacios, R. & Avendaño, O., 659 (Rab.)
Palanca, J. A., 952 (B.R.)
Palazuelos, P. G., with Alessandri & Lerner, 168 (Diet.)
Palestine, 538 (Pl.)
Palmer, A. E., Amolsch, A. L. & Shaffer, L. W., 330 (Der.)
Pampana, E., 427, 429 (Mal.)
Pan, C. S., 548 (Lep.)
Panja, G., 241 (Chl.)
—— & Ghosh, S. K., 910 (Chl.)
——, Malik, K. S. & Paul, B. M., 140 (Chl.)
—— & Paul, B. M., 909 (Chl.)
Pant, K. C. & Ray, H. N., (54) (Am.)
Panthier, R., with Giroud, 35 *bis*, 764 (Typh.)
Pape, with David, 19 (Tryp.)
Pará, M., with Fox, Manso & Penna, 41 (Y.F.)
Paracampos, H., with Macchiavello, 48, 49 *bis*, 50 (Pl.)
Parada Gay, M. A., with Varela, 532 (Fev.)
Pardo-Castello, V., Leon, E. R. & Trespalacios, F., 174 (Der.)
—— & Tiant, F. R., 785 (Lep.)
Parker, R. R., 448 (Fev.)
——, Kohls, G. M. & Steinhaus, E. A., 835 (Fev.)
—— & Steinhaus, E. A., 691 (Typh.), 769 (Fev.)
——, with ——, 691 (Typh.)
Parker, W. V., with Robertson, LePrince & Johnson, 15 (Mal.)
Parrot, L., 590 (Leish.), (871) (Misc.)
——, Catanei, A. & Ambialet, R., 739 (Mal.)
——, Donatien, A. & Plantureux, E., 527 (Leish.)
——, with Foley, 941 (Oph.)
Parsons, R. J., 331 (Der.)
Pasternack, J. G., 708 (Hel.)
Paterson, R. L., 865 (Misc.)
Patiño-Camargo, L., 298, 533, 900 (Typh.)
Patton, E. W., with Youmans, Robinson & Kern, 620 (Diet.)
Paul, B. M., with Panja, 909 (Chl.)
——, with —— & Malik, 140 (Chl.)
de Paula, H., (63) (Hel.)
Paulley, J. W., 241 (Dys.)
Pearce, A. H. B., 512 (Mal.)
Pearman, R. O. & Haseman, L., (724) (Misc.)
Pearse, A. S., 96 (B.R.)
Pédoya, C., (566) (Misc.)
Peel, E., with van Hoof & Henrard, 367, 368 (Tryp.)
Peláez Redondo, J., 375 (Leish.)
Pelazzi, A., 287 (Mal.)
Pellicano, V. L., with Brill, 819 (Mal.)
Pelner, L. & Saskin, E., 290 (Mal.)
Penido, H. M., with Causey & Deane, 815 (Mal.)
Penna, H. A., with Fox, Manso & Pará, 41 (Y.F.)
Penna, H. S. & Bittencourt, A., 840 (Y.F.)
Penna Sobrinho, O., 938 (Vms.)
Perez, B., (926) (Hel.)
Perez Acosta, F., 289 (Mal.)
Pérez Gallardo, F., with Clavero, 530 (Typh.), 641 (B.R.)
——, with Clavero del Campo, 952 (B.R.)
Perfiliev, P. P. & Poldoljan, V. J., 45 (Fev.)
Pergola, A., 81 (Vms.), (494) (Oph.)
Perinetti, H., with Rivas, (66) (Hel.)
Perrin, T. G., 711 (Hel.)
Perves, 155 (Lep.)
Pescott, R. T. M., 805 (Misc.)
Pessôa, S. B., 266 (Misc.)
—— & Coutinho, J. O., 762 (Leish.)
Pestel, M., with Brulé, 722 (Misc.)
Peter, F. M., 13 (Mal.)
Peter, H., with Gildemeister, 895 (Typh.)
Peters, H. T., 749 (Mal.)
Peterson, H. & Kõivastik, T., 624 (Vms.)
Petrov, M. I., 326 (Hel.)
Philip, C. B., 533, 828 (Fev.)
Piccinelli, A., 217 (Mal.)
Pickens, E. G., with Brigham, 690 (Typh.)
Pickhan, 766 (Typh.)
Pieraerts, G., 406 (Haem.)
Pinkerton, H., 25 (Typh.)
Pinkus, H., 863 (Der.)
Pinto, C. & Clausell, D. T., 579 (Mal.)
Piringer, W., 205 (Rab.)
Pirosky, I., Pirosky, R. de & Casiraghi, J. C. 474 (Hel.)

Pirosky, I., Sampayo, R. & Franceschi, C., 266 (Vms.)
Pirosky, R. de., with Pirosky, I. & de Casiraghi, 474 (Hel.)
Placeo, F., 869 (Misc.)
Plantureux, E., with Parrot & Donatien, 527 (Leish.)
Platel, G. & Vandergoten, Y., 177 (Misc.)
Plotz, H., 443 (Typh.)
——, Smadel, J. E., Anderson, T. F. & Chambers, L. A., 685 (Typh.)
Podoljan, V. J., with Perfiliev, 45 (Fev.)
Podyapolskaya, V. P., 474 (Hel.)
—— & Kamalova, A. G., 474 (Hel.)
Pogodina, E. A. & Sokolov, A. G., 15 (Mal.)
Pohlmann, E., 328 (Hel.)
Poirier, M. & Blondel, P., 848 (Hel.)
Poleff, L., 492 (Oph.)
Pollard, M., with Reynolds, 897 (Typh.)
Pollitzer, R. & Li, C. C., 776 (Pl.)
Pomales-Lebrón, A. & Morales-Otero, P., 528 (Typh.)
Pons, R., 776 (Pl.)
Poole, L. T., 52 (Dys.)
Pop, A., with Combiescu, Zotta, Maniculescu & Tascau, 34 (Typh.)
Popov, P. P. & Achundov, J. A., 314 (R.F.)
Popov, V. M., 434 (Mal.)
Porter, R. J., 221 (Mal.)
Pospelova-Shtrom, M. V., 544 (R.F.)
Potapchik, J. A., 893 (Typh.)
Potenza, L. & Andeze, P. J., 891 (Leish.)
Poth, E. J., Chenoweth, B. M., Jr. & Knotts, F. L., 396 (Dys.)
Power, M. H., with Williams, Mason & Wilder, 558 (Diet.)
Prendel, A. R., 521 (Mal.)
Price, A. H., with Reimann & Herbut, 853 (Hel.)
Priest, W. M., with Bulmer, 395, 777 (Dys.)
Prieto Casanova, J. T., 721 (Misc.)
Primitivo de la Quintana, 126 (Typh.)
Prince, F. M., 775 (Pl.)
Pritam Singh, with Covell, 663 (Mal.)
Proceedings of the New Jersey Mosquito Extermination Association, 335 (Misc.)
Prunes, L. & Frey, J. R., 491 (Der.)
Purchase, H. S., 363 (Mal.)
Putman, P., with Kitchen, 109 (Mal.)

Q.

Quinby, G. E., with Bang & Simpson, T. W., 817 (Mal.)

R.

Rachou, R. G., 514 (Mal.)
Račić, J., 792 (Hel.)
Radičev, with Mihaljevič, 446 (Typh.)
Radna, R. & Limbos, P., 316 (R.F.)
Raffaele, G. & Sandicchi, G., 218 (Mal.)
Rajagopal, M. D., with Naidu & Rao, 290 (Mal.)
Rames, C., 494 (Misc.)
Ramoo, H., with Rao, 114, 115 (Mal.)
Ramos, A. S., with Corrêa, 513 *bis* (mal.)
——, with Unti, 514 (Mal.)
Randolph, T. G., with Gibson, E. B., 860 (Haem.)
Ranta, L. E. & Dolman, C. E., 540 (Chl.)
Rao, A. V., with Naidu & Rajagopal, 290 (Mal.)
Rao, H. R., with Russell & Knipe, 114, 753 (Mal.)
Rao, P. J., with Rao, V. V. & Roy, 9 (Mal.)
Rao, R. B. & Ramoo, H., 114, 115 (Mal.)
Rao, S. R., 256 (Hel.)
Rao, S. S., 160 (Hel.)
Rao, T. R., with Russell, 8, 288, 289, 435 (Mal.)
——, with —— & Knipe, 14 (Mal.)
Rao, V. V., 113 (Mal.)
——, Roy, B. B. & Rao, P. J., 9 (Mal.)
Rappaport, I., 933, 934 (Hel.)
Rath, R. C., with Verghese, 546 (Lep.)
Ray, H. N., with Pant, (54) (Am.)
Ray, J. C., Mukerjee, S. & Roy, A. N., 118 (Mal.)
——, with Niyogi 762 (Leish.)
Ray, L. F. & Rockwood, E. M., 173 (Der.)
Reardon, L. V., with Rees, Bozicevich & Jones, 456 (Am.)
Rector, L. E., 927 (Hel.)
Reenstierna, J., 152 (Lep.)
Rees, C. W., Bozicevich, J., Reardon, L. V. & Jones, F., 456 (Am.)
Reichs-Gesundheitsblatt, 392 (Chl.)
Reid, R. D., with Findlay & Maegraith, 593 (Typh.)
Reimann, H., 258 (Hel.)
Reimann, H. A., Price, A. H. & Herbut, P. A., 853 (Hel.)
Reiss, F., 862 (Der.)
Reitler, R. & Btesh, S., 333 (Misc.)
Remlinger, P. & Bailly, J., 195, 196, 197, 198, 199, 653 *bis*. (Rab.)
Rémond, M., 443 (Typh.)
Renn, C. E., 582 (Mal.)
Renoux, G., 894 (Typh.)
Resseler, R., (267) (Misc.)
Rey, F., 442 (Leish.)
Reynolds, F. H. K. & Pollard, M., 897 (Typh.)
Reznikoff, P., with Kauer, Jr. & Bird, 560 (Vms.)
Ricardo Benavente, G., (254) (Hel.)
Rice, M. E. & Watson, R. B., 819 (Mal.)
——, with —— & Maher, 665 (Mal.)
Richard, H., with Sergent, (918) (R.F.)
Richards, A. G., Jr., 90 (Misc.)
Richards, H., 499 (Misc.)
Richardson, A. P., with Hewitt & Seager, 439 (Mal.)
Richardson, D. T., 631 (Misc.)
Riding, D., 540 (Dys.)
Riesel, M. A., with Savino, 774 (Pl.)
——, with Sordelli, Manzullo & Ferrari, 385 (Typh.)
Rigdon, R. H., 436 (Mal.)
—— & Thomas, W. K. S., 118 (Mal.)
Riley, E. G., 793 (Hel.)
Riordan, T. J., Gellis, S. & Rubinowitz, A. M. 859 (Diet.)
Rissmann, E. F., 147 (Am.)
Rivas, C. I. & Perinetti, H., (66) (Hel.)
Roberts, G. H., with Hampil, 200 (Rab.)
Roberts, H. R., with Ross, 877 (Mal.)

Robertson, J. L., Jr., LePrince, J. A., Johnson, H. A. & Parker, W. V., 15 (Mal.)
Robertson, P. A. M., (313) (Am.)
Robic, J., with Girard, 777 (Pl.)
Robinson, G. G., 243, (244) (R.F.)
Robinson, P., 686 (Typh.)
Robinson, W. D., with Youmans, Patton & Kern, 620 (Diet.)
Rocha, A., with de Magalhães, (136), 300 (Typh.)
Rockefeller Foundation, 112 (Mal.), 340, (343) (B.R.)
Rockwood, E. M., with Ray, 173 (Der.)
Roda, E., with Jiménez-Diaz, Ortiz de Landázuri, Marina & Lorente, 87 (Misc.)
Rodaniche, E. C. & Kirsner, J. B., 455 (Am.)
Rodhain, J., 167 (Hel.), 438 (Mal.)
—— & van Hoof, M. T., 214 (Mal.)
—— & Lassman, P., 119 (Mal.)
——, Valcke, G. & van Goidsenhoven, C., 120 (Tryp.)
Rodriguez Olleros, A., (170) (Sp.)
Röpke, F., 868 (Misc.)
Rose, 832 (Typh.)
Rose, H. M., with Culbertson, 471 (Hel.)
Rosenberger, R. C., with Smith, (181) (Misc.)
Rosenthal, S. M., 589 (Tryp.)
Ross, E. S. & Roberts, H. R., 877 (Mal.)
Ross, H., with Faget & Johansen, 610 (Lep.)
Rossas, T. P., 150 (Lep.)
Rossi, G., 927 (Hel.)
Rossier, P. H. & Dressler, M., 700 (Am.)
Rost, G. S., 608 (Y. & S.)
Rotenburg, S. S., 300 (Typh.)
Roubaud, E., Chorine, V. & Guiraud, P., 109 (Mal.)
Row, R. & Kulkarni, S. S., 378 (Leish.)
Roy, A. N., with Niyogi, 755 (Mal.)
——, with Ray & Mukerjee, 118 (Mal.)
Roy, B. B., with Rao, V. V. & Rao, P. J., 9 (Mal.)
Roy, D. N. & Biswas, T. C., 112 (Mal.)
—— & Ganguli, S. K., 749 (Mal.)
—— & Ghosh, S. M., 413 (Misc.)
Rubiano Groot, H., 861 (Haem.)
Rubinowitz, A. M., with Riordan & Gellis, 859 (Diet.)
Ruchman, I., with Sabin, 636 (Misc.)
Russell, P. F., 882 (Mal.)
—— & Jacob, V. P., 105 (Mal.)
——, Knipe, F. W. & Rao, H. R., 114, 753 (Mal.)
——, —— & Rao, T. R., 14, 114 (Mal.)
——, with ——, 115, 673 (Mal.)
—— & Menon, P. B., 362 (Mal.)
—— & Mohan, B. N., (17), 362 (Mal.)
——, Mulligan, H. W. & Mohan, B. N., 117 (Mal.)
—— & Rao, T. R., 8, 288, 289, 435 (Mal.)
Russo, E., with Coutinho, 174 (Der.)
Rutishauser, A., 72 (Hel.)
Ryan, E., with Boehrer & Stanford, 619 (Diet.)
Ryžov, A. A., 765 (Typh.)

S.

Sabin, A. B., 637 (Misc.)
—— & Ruchman, I., 636 (Misc.)
—— & Warren, J., 638 (Misc.)
——, with ——, 637, 638 (Misc.)
Sacks, S., with Javett, 216 (Mal.)
Sadusk, J. F., Jr. & Oswald, E., 843 (Chl.)
Salazar Mallen, M., with Millan Gutierrez & Martinez Baez, M., 482 (Diet.)
Saliternik, Z., 287 (Mal.)
Sampayo, R., with Pirosky & Franceschi, 266 (Vms.)
Samwick, A. A., with Morrison & Landsberg, 407 (Haem.)
Sandicchi, G., with Raffaele, 218 (Mal.)
Sanyal, S. & Maitra, M. N., 492 (Oph.)
Sapero, J. J., Hakansson, E. G. & Louttit, C. M., 53 (Am.)
Sarkar, B. B., Maitra, S. R. & Ghosh, B. N., 265 (Vms.)
Sarkar, S. N., 624 (Diet.)
Sarkissian, M., with Matevossian & Markaryan, 310 (Am.)
Sarles, M. P., with Taliaferro, 319 (Hel.)
Sarma, M. L., with Khorana & Giri, 80 (Diet.)
Saskin, E., with Pelner, 290 (Mal.)
Sati, M. H., with Kirk, 684 (Leish.)
Satulsky, E. M., 488 (Der.)
—— & Wirts, C. A., 863 (Der.)
Sautet, J., 110 (Mal.), 461 (R.F.)
Savage, T. R., 844 (Dys.)
Savateev, N. I., 911 (Am.)
Savino, E. & Riesel, M. A., 774 (Pl.)
—— & Villazón, N. M., 306 (Pl.)
Scarborough, H., 78 (Diet.)
Schaeffer, M. & Leider, A. G., 199 (Rab.)
Schäfer, W., 600 (Typh.)
Scharff, J. W., 640 (Misc.)
Scheinker, I. M., 690 (Typh.)
Scheller, E., 299 (Typh.)
Schenken, J. L. & Moss, E. S., 555 (Hel.)
Schill, E., 790 (Hel.)
Schmidt, L. H., with Mills, 567 (Misc.)
Schneider, K., 245 (Lep.)
Schoenaers, F., with van Goidsenhoven, 758 (Tryp..)
Schoenbach, E. B. & Spingarn, C. L., 359 (Mal.)
Schoetter, M., with Devignat, 391 (Pl.)
Schöttler, W. H. A., 560, 626 (Vms.)
Schramm, E., 545 (Y. & S.)
Schujman, S. & Vaccaro, A., 547 (Lep.)
Schütz, F. & Messerschmidt, T., 132 (Typh.)
Schwarz, E., 240 (Pl.)
Schwetz, J., 7, 102, 104, 212 (Mal.)
——, & Baumann, H., 103, 211 *bis* (Mal.)
——, with ——, 212 (Mal.)
——, with ——, Beumer & Fort, 213, 357 (Mal.)
——, —— & Fort, M., 743 (Mal.)
—— & Dartevelle, E., 249 (Hel.)
Science, 290 (Mal.)
Scops, C., 250 (Hel.)
Scotcher, N. M., with Wien & Freeman, 683 (Leish.)
Scott, J. A., 549, 553 (Hel.)
Scull, J. M. L., with Cabrera Calderín & Barreras, 262 (Hel.)

Seager, L. D., with Hewitt & Richardson, 439 (Mal.)
Seeler, A. O., Dusenbery, E. & Malanga, C., 879 (Mal.)
——, Graessle, O. & Dusenbery, E. D., 756 (Mal.)
Seitz, W., 859 (Diet.)
Semadeni, B., 616 (Hel.)
Semenova, N. E., 871 (Misc.)
Sen, G. N., with Napier & Sen Gupta, 121 (Leish.)
Senekji, H. A., 681 (Tryp.), 685 (Leish.)
Senevet, G. & Abonnenc, E., (870) (Misc.)
——, Chabelard, R. & Abonnenc, E., (870) (Misc.)
——, Moutier, P., Alcay, L., Gros, G. & Bourgarel, R., 805 (Misc.)
Sen Gupta, P. C., 359 (Mal.)
——, with Napier & Sen, 121 (Leish.)
Senra, J. & Felicissimo, O., 550 (Hel.)
Sergent, A. & Richard, H., (918) (R.F.)
Sergent, E., 180 (Misc.), (433) (Mal.), 485 *quat.*, 486, (486) (Vms.)
Shaffer, L. W., with Palmer & Amolsch, 330 (Der.)
Shannon, R. C., 522 (Mal.)
Shapkin, L. A., 437 (Mal.)
Shattuck, G. C. & Mixter, W. J., 953 (B.R.)
Shee, J. C., 604 (Fev.)
Shehadi, W. H., with Yenikomshian, 614 (Hel.)
Sheldon, A. J. & Groover, M. E., Jr., 321 (Hel.)
Shelley, H. M., (621) (Diet.)
Sherrard, G. C., 842 (Pl.)
Shih Lu Chang, 310 (Am.)
Shikhobalova, N. P., (168) (Hel.)
Shlenova, M. F., 520 (Mal.)
Shortt, H. E., with Menon & Ayyar, 220 (Mal.)
——, with Swaminath & Anderson, 227 (Leish.)
Shute, P. G., with Murray, 9, 821 (Mal.)
——, with Sinton, 413 (Misc.)
——, with Whelen, 752 (Mal.)
Sicault & Messerlin, (284) (Mal.)
Siddons, L. B., with Mukerji & Ghosh, 670 (Mal.)
Siedek, H., 143 (Dys.)
——, with Fanta, 234 (Typh.)
Siegfried, G. & Izac, R., 105, 738 (Mal.)
Sierra Leone, Medical Department, 432 (Mal.)
Sieyro, L., 698 (Am.)
Sigalos, P., 864 (Der.)
Sikora, H., 763 (Typh.)
Silverberg, M. G. & Henschel, E. J., 377 (Leish.)
Simeons, A. T. W., 667 (Mal.)
Simmons, J. S., 283 (Mal.)
—— & Aitken, T. H. G., 954 (B.R.)
Simões, M. P., with Hill & Cambournac, 818 (Mal.)
Simonetti, G., 25 (Leish.)
Simpson, T. W., with Bang & Quinby, 817 (Mal.)
Simpson, T., with Bang, 359 (Mal.)
Simson, F. W. & Barnetson, J., 176 (Der.)
——, Harington, C. & Barnetson, J., 630 (Der.)
Singh, B., 901 (Typh.)
Sinton, J. A. & Shute, P. G., 413 (Misc.)
Sisk, W. N., 927 (Hel.)
Sitapathy, N. R., with Knipe, 116 (Mal.)
Sivalingam, V., 746 (Mal.)
Skeer, J., 490 (Der.)
Skoog, A. P., with Marble & Bucholz, 258 (Hel.)
Skopin, I. G., 287 (Mal.)
Skrjabin, K. I. & Mathevossian, E. M., (400) *ter* (Hel.)
Skvortzov, A. A. & Talysin, F. F., 158 (Hel.)
Slack, R., with Barber & Wien, 376 (Leish.)
Smadel, J. E., with Plotz, Anderson & Chambers, 685 (Typh.)
Smarr, R. G., with Young & McKendon, 881 (Mal.)
Smart, A. G. H., 631 (Misc.)
Smith, C. D. & Rosenberger, R. C., (181) (Misc.)
Smith, D. C., with Kaufman, 483 (Sp.)
Smith, D. J. W., 388 (Typh.), 601 (Fev.)
——, with Derrick & Brown, 302 (Fev.)
Smith, E. C., 496 (Misc.)
—— & Howie, J. W., 303 (Y.F.)
Smith, F. & Evans, R. W., 384 (Typh.), 523 (Bl.)
Smith, H., 631 (Misc.)
Smith, J. F., 25 (Leish.)
Smith, J. V., 215 (Mal.)
Smith, S. J., with Goldmann, 919 (Y. & S.)
Smyly, H. J., 540 (Dys.)
Smyth, C. J., Finkelstein, M. B., Gould, S. E., Koppa, T. M. & Leeder, F. S., 780 (Dys.)
Soap, New York, 882 (Mal.) 899 (Typh.)
Sobotka, H., with Adlersberg, 621 (Sp.)
Sofiev, M., 314 (R.F.)
Sokolov, A. G., with Pogodina, 15 (Mal.)
Solomon, H. C., with Kopp, 582 (Mal.)
Sonnenschein, 125 (Typh.)
Sonnenschein, C., 84, 95 (Misc.), 529 (Typh.)
Soper, F. L., 536 (Y.F.)
—— & Wilson, D. B., 670 (Mal.)
Sordelli, A., Manzullo, A., Riesel, M. A. & Ferrari, J., 385 (Typh.)
Soricelli, F., (179) (Misc.)
South African Institute for Medical Research Annual Report, 179 (Misc.)
South African Medical Journal, 865 (Misc.)
Spadaro, O., 908 (Fev.)
Spaeth, H., 258, 478 (Hel.)
Spencer, G. A., 85 (Misc.)
Spingarn, C. L., with Schoenbach, 359 (Mal.)
Stabile, A., (567) (Misc.)
Stage, H. H. & Yates, W. W., 335 (Misc.)
Stanford, C. E., with Boehrer & Ryan, 619 (Diet.)
Stanley-Jones, D. & Harris, C. E. S., 81 (Vms.)
Stannus, H. S., 259 (Sp.)
Stasney, J., 623 (Haem.)
Stefanopoulo, G., 852 (Hel.)
Steigman, A. J., with van den Ende, Stuart-Harris & Harries, 449 (Typh.)
Stein, H. J., with Follis, Jr., Miller, M. H. & Wintrobe, 795 (Diet.)
Steinhaus, E. A., 238 (Fev.)
—— & Parker, R. R., 691 (Typh.)
——, with ——, 769 (Fev.)
——, with —— & Kohls, 835 (Fev.)
——, with ——, 691 (Typh.)

Stephenson, R. W., 821 (Mal.)
Stern, K. & Dancey, T. E., (935) (Hel.)
Steuer, W., 898 (Typh.)
Stewart, M. A., 306, 841 (Pl.)
—— with Hosford & Sugarman, 257 (Hel.)
Stockdale, F., 632 (Misc.)
Stone, R. G., 806 (Misc.)
Stowens, D., 403 (Hel.)
Strangeways, W. I., with King, 20 (Tryp.)
Stratman-Thomas, W. K., with Dulaney & Warr, 11 (Mal.)
——, with Knisely & Eliot, 584 (Mal.)
Strong, R. P., 953 (B.R.)
Stshensnovitsh, V., 313 (Am.)
Stuart-Harris, C. H., with van den Ende, Harries & Steigman, 449 (Typh.)
Sturm, A., 32, 381 (Typh.)
Suàrez, R. M., with Díaz Rivera & Hernández Morales, (267) (Misc.)
Sugarman, E. I., with Hosford & Stewart, 257 (Hel.)
Sukhareva, N. D., with Melnikov & Fedder, 90 (Misc.)
Sullivan, W. N., Goodhue, L. D. & Fales, J. H., 337 (Misc.)
——, with McGovran, 338 (Misc.)
Summers, W. A., (709) (Hel.)
—— & Weinstein, P. P., 924 (Hel.)
Sundermann, A., with Mumme, 328 (Hel.)
Süpfle, K. & Fischer, H., 688 (Typh.)
Sútter, V. A. & Zúniga, H., 107 (Mal.)
Suzman, M. M., (796) (Diet.)
Swaminath, C. S., Shortt, H. E. & Anderson, L. A. P., 227 (Leish.)
Sweet, W. C., Feng, L. C., Chow, C. Y. & Hsu, S. C., 666 (Mal.)
Swyer, R., 844 (Dys.)
Sylla, A., 38 (Fev.), 445 (Typh.)

T.

Taiana, J. A., with Brea & Niño, 698 (Am.)
Taliaferro, W. H. & Sarles, M. P., 319 (Hel.)
Tallman, E., (66) (Hel.)
Talysin, F. F., with Naidenova, (173) (Vms.)
——, with Skvortzov, 158 (Hel.)
Tanganyika Territory, Med. Dept., 879 (Mal.)
Tareyev, E. M., 668 (Mal.)
Tascau, J., with Combiescu, Zotta, Manciulescu & Pop, 34 (Typh.)
Tassi, G., 856 (Diet.)
Taylor, F. H., 870 (Misc.)
Taylor, H. G., with Franks, 940 (Der.)
Taylor, J., with Ormiston & Wilson, 54 (Am.)
Tenner, K., 94 (Misc.)
Tey, A., Criscuolo, E. & Marhuenda, P., 333 (Misc.)
Thomas, R. H., 799 (Misc.)
Thomas, W. K. S., with Rigdon, 118 (Mal.)
Thompson, K., 821 (Mal.)
Thomson, R. C. M., 436, 747, 748 (Mal.)
Tiant, F. R., with Pardo-Castello, 785 (Lep.)
Tietz & Carlé, 133 (Typh.)
Tobias, J. M., 865 (Misc.)
Tomita, M., 113 (Mal.)
Tommaso, P., 880 (Mal.)
Tompsett, R., with McDermott, Webster, Baker & Lockhart, 679 (Tryp.)
Tong, W. K., with Chang, Li & Chin, 851 (Hel.)
Tonkin, A. H., 865 *bis* (Misc.)
Topping, N. H., 837 (Fev.)
—— & Dyer, R. E., 690 (Typh.)
Toranzos, L. B., Figuerero, M. & Barbará, L., 680 (Tryp.)
Torrealba, J. F., 680 (Tryp.)
Torres Estrada, A., 709 (Hel.)
Townsend, R. F., with de Saram, 820 (Mal.)
Trager, W., (584), 675, 676, 825 (Mal.)
Trant, H., 154 (Lep.)
Trautmann, 383 (Typh.)
Travassos, J. & Vallejo, A., 386, 387 (Fev.)
—— & Vallejo-Freire, A., 768 (Typh.)
Treillard, M., (667) (Mal.)
Trespalacios, F., with Pardo-Castello & Leon, 174 (Der.)
Trethewie, E. R., 409 (Vms.)
Treu, R., 720 (Misc.)
Trimarchi, M., (56) (R.F.)
Trolli, G., 178 (Misc.), 213 (Mal.)
—— & van Wymeersch, 18 (Tryp.)
Trowell, H. C., 329, 405, 484, 938 (Haem.)
Tshalaia, L., 311 (Am.)
Turnbull, F. M. & Franklin, L. B., 340 (Misc.)
Turner, E. A., with Andrews & Howard, 673 (Mal.)
Turner, P. E., 875 (B.R.)

U.

Udenfriend, S., with Brodie, 821 (Mal.)
Unti, O., 515 *bis* (Mal.)
—— & Ramos, A. S., 514 (Mal.)
Uttley, K. H., (96) (Misc.)

V.

Vaccaro, A., with Schujman, 547 (Lep.)
Vakil, R. J., 783 (R.F.)
Valcke, G., with Dubois, (789) (Lep.)
——, with Rodhain & van Goidsenhoven, 120 (Tryp.)
Valencia Parparcén, J., (613) (Hel.)
Vallejo, A., with Travassos, 386, 387 (Fev).
Vallejo-Freire, A., with Travassos, 768 (Typh.)
Van den Berghe, L., 40 (Y.F.) 162 *ter* (Hel.) 262 (Haem.)
Van den Ende, M., Stuart-Harris, C. H., Harries, E. H. R. & Steigman, A. J., 449 (Typh.)
Vandergoten, Y., with Platel, 177 (Misc.)
van Goidsenhoven, C., with Rodhain & Valcke, 120 (Tryp.)
—— & Schoenaers, 758 (Tryp.)
van Hoof, L., Henrard, C. & Peel, E., 367, 368 (Tryp.)
van Hoof, M. T., with Rodhain, 214 (Mal.)
van Meerendonk, P., 237 (Typh.)
van Riel, J., 462 (R.F.)
van Rooyen, C. E. & Bearcroft, W. G. C., 895 (Typh.)
van Studdiford, M. T., 797 (Der.)
van Wymeersch, H., 213 (Mal.)
—— with Trolli, 18 (Tryp.)

Varela, G., with Mazzotti, 301 (Typh.)
—— & Parada Gay, M. A., 532 (Fev.)
Vargas, L., 74 (Hel.)
Vassilkova, Z., 318 *bis* (Hel.)
Vaucel, 586 (Tryp.)
Vaughn, L. D., with Broders, Dochat & Herrell, 798 (Der.)
Vaux, D. M., 483 (Sp.)
Vedder, E. B., 482 (Sp.) 557 (Diet.)
Vellard, J., 408 (Vms.)
Vendrely, R., with Boquet, 862 (Vms.)
Venkataraman, K. V., 139 (Chl.)
Venzant Quintana, E., 400 (Hel.)
Verghese, G. & Rath, R. C., 546 (Lep.)
Verma, O. P., 492 (Oph.)
——, with Aykroyd, 491 (Oph.)
Versiani, O. & Junqueira, M., 890 (Tryp.)
Viala, C., with Cruveilhier, 203 (Rab.)
Viana Martins, A. & Macedo, E., 761 (Tryp.)
Videla, C. A., 13 (Mal.)
Vieira, G., 797 (Der.)
Vieira, J. P., 488 (Der.)
Vilanova, J., 828 (Leish.)
Villazón, N. M., with Savino, 306 (Pl.)
Vilén, A. F., 801 (Misc.)
Vincke, I. & Janssens, P. G., 391 (Pl.)
Visweswar, with Heilig, 360 (Mal.), 400 (Hel.), 406 (Haem.)
Vivanco, F., with Jiménez-Diaz, 87 (Misc.)
Vogt, P., 528 (Leish.)
Volk, R. & Canas, E., 410 (Der.)

W.

Walker, A. J. & Davey, T. H., 432 (Mal.)
Walker, A. S., Meyers, E., Woodhill, A. R. & McCulloch, R. N., (604) (Fev.)
Walker, J. W. & Charters, A. D., 869 (Misc.)
Walker, N. F., 862 (Vms.)
Walther, G., 393 (Dys.)
—— & Günther, L., 51 (Dys.)
Wang, C. W., with Chung & Lee, 229 (Leish.)
Wanson, M., 414 (Misc.)
War Medicine, Chicago, 943 (Misc.)
Warnecke, B., 689 (Typh.)
Warner, B. W., 155 (Hel.)
War Office, 343 (B.R.), 900 (Typh.)
War Office. Army Medical Department Bulletin, 268 (Misc.)
Warr, O. S., with Dulaney & Stratman-Thomas, 11 (Mal.)
Warren, J. & Sabin, A. B., 637, 638 (Misc.)
Watson, R. B., Maher, H. C. & Rice, M. E., 665 (Mal.)
——, with Rice, 819 (Mal.)
Watt, J. Y. C., 792 (Hel.)
——, Golden, W. R. C., Olason, F. & Mladinich, G., 793 (Hel.)
Watt, J., with Brigham, 238 (Typh.)
Webb, B. D., with Andrews, 158 (Hel.)
Webster, B., with McDermott, Baker, Lockhart & Tompsett, 679 (Tryp.)
Webster, L. T., 269 (B.R.)
—— & Casals, J., 201 *bis* (Rab.)
Weed, L. H., 361 (Mal.)
Weil, A. J., 543 (Dys.)
Weiner, A. L., Gaynon, I. E. & Osherwitz, M. S., 493 (Oph.)
Weingarten, R. J., 407 (Haem.)
Weinstein, P. P., with Summers, 924 (Hel.)
Weise, E. C., 490 (Der.)
Weiss, P., 769, 770 (Bart.)
Weller, T. H., 852 (Hel.)
Wendel, W. B., 674 (Mal.)
Wenschr, 382 (Typh.)
Werner, H., 387 (Fev.)
Weselmann, H., 606 (Am.)
West, R. F., 779 (Dys.)
Westphal, A., 144, 147 (Am.)
Westphal, K., 692 (Fev.)
Westphal, R. S., (794) (Hel.)
Wheeler, C. M., (545) (R.F.)
——, with Douglas, 695 (Pl.)
——, with Evans & Douglas, 696 (Pl.)
Whelen, M. & Shute, P. G., 752 (Mal.)
Whildin, J., with Arnold, 492 (Oph.), 941 (Der.)
Whitby, L. E. H. & Britton, C. J. C., 503 (B.R.)
White, H. J., with Marshall & White, 223 (Mal.)
White, T. H., 869 (Misc.)
Whitman, L., 534 (Y.F.)
Whitteridge, S. M., 329 (Hel.)
Wickremesinghe, W. F., 854 (Diet.)
Wien, R., 682 (Leish.)
——, with Barber & Slack, 376 (Leish.)
——, Freeman, W. & Scotcher, N. M., 683 (Leish.)
Wigglesworth, V. B., 89 (Misc.)
Wijesundara, D. P., 804 (Misc.)
Wilcox, A., 432 (Mal.)
Wilder, R. M., with Williams, Mason & Power, 558 (Diet.)
Wiley, A. J., with Lewis & Macaulay, 887 (Tryp.)
Wilkins, E. G., 67 (Hel.)
Wilkinson, P. B., 843 (Chl.)
Williams, R. D., Mason, H. L., Power, M. H. & Wilder, R. M., 558 (Diet.)
Wilson, C., with Blacklock, 358 *bis*. (Mal.)
Wilson, C. M., 417 (B.R.)
Wilson, D. B., with Soper, 670 (Mal.)
Wilson, D. C., 394 (Dys.)
Wilson, G. S., with Ormiston & Taylor, 54 (Am.)
Wilson, H. T. H. & Ellis-Jones, D. W., 723 (Misc.)
Windorfer, A., 446 (Fev.)
Winfield, G. F., with Meng, 871 (Misc.)
Wingfield, A., 518, 821 (Mal.)
Wintrobe, M. M., with Follis, Jr., Miller & Stein, 795 (Diet.)
Wirts, C. A., with Satulsky, 863 (Der.)
Wohlrab, R., (32) (Typh.)
Wolf, A., with Cowen & Paige, 950 (Misc.)
Wolfe, H. R. I., 850 (Hel.)
Wood, S. F., 525 (Tryp.)
Woodhill, A. R., with Walker, Meyers & McCulloch, (604) (Fev.)
Wright, A. A., 945 (Misc.)

Wright, A. M., with Co Tui, 140 (Chl.)
Wright, W. H. & Bozicevich, J., (854) (Hel.)

Y.

Yarwood, G. R. & Elmes, B. G. T., 704 (Hel.)
Yates, W. W., with Stage, 335 (Misc.)
Yen, C. H., 204 (Rab.)
Yenikomshian, H. A. & Shehadi, W. H., 614 (Hel.)
Yonis, Z., 913 (Am.)
Yorke, W., with Fulton, 754 (Mal.)
——, with —— 19 (Tryp.), 23 (Leish.)
Youmans, J. B., Patton, E. W., Robinson, W. D. & Kern, R., 620 (Diet.)
Young, C. T., Cook, W. R. & Kawasaki, I. A., 635 (Misc.)
Young, M. D., McKendon, S. B. & Smart, R. G., 881 (Mal.)
Young, W. A. & Gordon, S., 73 (Hel.)

Z.

Zanetti, V., with Degotte, 495 (Misc.)
Zaturian, A., 312 (Am.)
Zak, O. S., with Bronshtein, 661 (Rab.)
Zemp, F. E., 602 (Fev.)
Zimmermann, E., 27 (Typh.)
Zotta, G., with Combiescu, Manciulescu, Pop & Tascau, 34 (Typh.)
Zumpt, F., 7 (Mal.)
Zúniga, H., with Kumm, 108 (Mal.)
——, with Sútter, 107 (Mal.)
Zvereva, N. S., with Dinnik, (66) (Hel.)

INDEX OF SUBJECTS.

(The entries in heavy type refer to Sections in which abstracts on the subject indicated are grouped together. Page numbers within brackets indicate papers cited by title only).

Actinomyces madurae, 630
mexicanus, causing mycetoma, 489
Actinomycosis in Greece, 864
Adder bite, in Britain, 81
Adenitis in Chagas's disease, 227, 890
in leprosy, 547
Aëdes spp.
in Salvador, 108
Sudan, 602
and *Anopheles maculipennis*, comparative fertility and longevity, 566
biology of, 565
control, 39
life history, 512
aegypti
in Australia, 870
Brazil, 670
Sudan, 602
atlas of, 877
control, in Brazil, 670
susceptibility to *P. lophurae*, (584)
furcifer, in Sudan, 602
luteocephalus, in Sudan, 602
metallicus, in Sudan, 602
simpsoni
breeding places of, 304
yellow fever and, in Uganda, 304
var. *lilii*, in Sudan, 602
taylori, in Sudan, 602
vexans, flight range of, 748
vittatus, in Sudan, 602
Age, determination of, in Bengali girls, use of X-ray of bones for, 181
Agranulocytosis following quinine therapy, (821)
Agriculture, malaria and, 753
Ainhum
in U.S.A., in negro, 85
hyperkeratosis palmaris et plantaris and, 85
Aircraft
insect control on, 908
rôle of, in disease transmission, 563
Amaurosis, toxic
due to quinine, 290
treatment
ethylmorphine hydrochloride, 290
sodium nitrite, 290
Ambassadors in White, Story of American Tropical Medicine [WILSON] (book review), 417
Amblyomma
americanum, transmitting Rocky Mountain fever, 534, 835
cayennense, transmitting Rocky Mountain fever, 534
Amblyopia, tryparsamide, and nutritional degeneration, 679
AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS, 52–54, 144–148, 243, 310–313, 455–460, 606, 697–700, 781–782, 911–916, see also *Entamoeba histolytica*

Amoebiasis and Intestinal Protozoal Infections —*cont.*
in Africa, South-West, 84
West, 945
Germany, 606, 781, 913
India, 606
Mexico, 455, 459
Natal, in children, 398
Nigeria, in children, 497
Palestine, 913
U.S.S.R., 312, 313
U.S.A., 697
Mississippi, 70
New Orleans, 53
Venezuela, 51
arthritis and, 913
in children, 398, 497
complications, 606
diagnosis
complement fixation test, 456
differential, from bacillary dysentery, 541
experimental, 144
liver abscess
in Argentine, 698
U.S.A., 243, 915
atypical, in Hindu woman, 916
diagnosis, 146, 458
treatment, 699, 916
aspiration, 147
emetine, 146, 243
lung abscess
in Argentine, 698
treatment, 699
of penis
in U.S.A., 457
treatment, 457
treatment
carbarsone, 181
diodoquin, 54, 697
emetine, 144, 606
and yatren, 913
"Wia," 913
Trichomonads, blood-ingesting, in, 459
urinary, treatment by emetine, 144
Amoebic tumours of large intestine, treatment, 145
Anaemia
in Nigeria, in children, 497
in avian malaria, causing death, 293
cholesterol and, 262
deficiency
in Uganda, 329, 484
morphology of blood in, 329
dimorphic, 938
due to combination of malaria and Necator infection, 431
macrocytic orthochromic, 405
pernicious, and sprue, similarities and differences between, (170)

Anaemia—*cont.*
sickle-cell
in Brazil, 171
Cuba, 714
Nigeria, in children, 497
U.S.A., 407, 623
abdominal crises in, 262
aetiology, 171
in children, 497
diagnosis, splenic puncture, 407
erythrophagocytosis and haemosiderosis in, 623
and familial haemolytic jaundice, similarity between, 714
symptoms, 714
treatment, 263
treatment, 406
tropical, 861
Ancylostoma, see under Hookworm
Aniacinosis, 559
in U.S.A., 169
tongue changes in, 169
treatment, 169
Ankylostomiasis *see also* Hookworm
in Africa, South West, 84
Bathurst Island, 412
Belgian Congo, 251
Brazil, São Paulo, 266
China, Szechwan, 851
Dutch East Indies, Java, 326
India, 68
Coorg, 170
Melville Island, 412
Mexico, Usúmacinta Valley, 453
Morocco, 708
Natal, in children, 398
Nigeria, in children, 497
Syria, 614
U.S.A., 324
Mississippi, 70
anaemia
in India, 68, 400, 406
iron-refractory, in India, 406
treatment, effect on gastric function, 400
in children, 398, 497
complications, cardiac, 68
control, 615
in U.S.A., Georgia, 324
duodenal ulcer syndrome, caused by, 614
Necator americanus infection
in Belgian Congo, 251
Brazil, 579
in children, 550
Dutch East Indies, Java, 326
acquired immunity, experiments, 321
Loeffler's infiltration in, 400
malaria and, anaemia due to, 431
prophylaxis, 615
treatment, 615
ascaridol, experiments, 62
E.1750, experiments, 62
430 K1, experiments, 60, 62
iron, 325
phenothiazine, 702
tetrachlorethylene, 325
quinine and, 158
Anopheles
of Albania, 286
Algeria, 494, 740
Anopheles—*cont.*
of Angola, 744
Argentine, 219, 745
Belgian Congo, 7, 357
Ruanda, 211
Belgium, 214
Brazil, 513, 514, 515, 746
British Guiana, 513
China, Yunnan, 666
Cuba, 745
Eritrea, (215)
Greece, 429
Guatemala, 292, (878)
India, 115, 219
Nilgiris, 106
Udaipur State, 113
Lebanon, 110
Mexico, (878)
Mozambique, 110, 111, 112
Nicaragua, 359
Palestine, 287
Porto Rico, 754
Salvador, 108
Syria, 110
Turkey, 816
U.S.A., Georgia, 215
U.S.S.R., 824
Yugoslavia, 429
breeding
effect of stocking ricefields with sullage, on 113
places of, 431
*p*H of, 515
collection and transportation, 579
complex, in U.S.A., California, (113)
control, 111, 670, 671, 944
in Algeria, 105
India, 673
on ricefields, 113, 114
Uganda, 672
U.S.S.R., 427, 824
in agricultural villages, 358
airplane in, 17
in airplanes, 564, 908
alkaloidal fogs, 15
automatic siphon, 674
disinsectization of day-time shelters, 521
ditch lining, 15, 673
drainage, 219, 673
fish breeding, 114, 430, 431, 437, 494, 673 741
flushing, (825)
irrigation, 673
of ricefields, 114
larvicides, 335, 741
calcium arsenite, 742, 753
copper arsenite, 17
cotton-seed tar, 672
distillate from coke stills, 521
freon-pyrethrum aerosol, 882
mineral oils, comparative suitability of, 1
Paris green, 219, 672, 673, 754
automatic distribution, 115
-lime mixtures, 16
petroleum, 219
use of waste lubricating oil in, 293
oiling, 219, 431, 741, 754
plant-growing on canal banks, 115
pyrethrum extract, 14

Anopheles—*cont.*
control—*cont.*
spraying, 116, 882
sullage on ricefields, 113
Xenopus clivii, 744
eggs
collection and transportation, 579
macroscopic differentiation of, on surface of water, 287
flight range of, from breeding places, 742
at high altitudes, 514
larval, species identification, macroscopic, 215
Nearctic, atlas, of, 877
Anopheles
aconitus
in India, 114
Nilgiris, 106
Udaipur State, 113
aitkeni, in India, Nilgiris, 106
albimanus, malaria and
in Cuba, 745
Guatemala, 292
Porto Rico, 754
Salvador, 108
albitarsis, breeding places of, 515
algeriensis, in Syria, 110
amictus
in Australia, 870
differentiation from *A. annulipes*, 870
annandalei var. *interruptus*, in India, Nilgiris, 106
annularis
in India, 106, 114
malaria and, in India, 114
annulipes
in Australia, 870
differentiation from *A. amictus*, 870
apimacula, in Salvador, 108
argyritarsis
in Grenada, (512)
Salvador, 108
breeding places of, 515
control, flushing, (825)
barbirostris, in India, 106, 113
bellator, naturally infected with malaria, 289
bifurcatus, 288
life history, 512
brunnipes, in Mozambique, 112
christyi, in Belgian Congo, 8
clarki, n. sp., 818
claviger, malaria and, in Lebanon, 110
coustani
in Mozambique, 112
breeding places of, measurement of temperature and light in, 816
var. *tenebrosus*, in Mozambique, 111
crucians, in Nicaragua, 359
cruzi
experimentally infected with *P. vivax*, 515
naturally infected with malaria, 289
culicifacies
in India, 8, 106, 113, 114
breeding, control of, 114
control, plant-growing on canal banks, 115
density of, in relation to malaria endemicity, 289
habits of, 8
longevity of, 288
malaria and, in India, 219, 673
Anopheles—*cont.*
culicifacies—*cont.*
ovipositing, relation of mechanical obstruction and shade to, 435
darlingi
in Brazil, São Paulo, 267
Mexico, Usumacinta Valley, 453
eggs, characteristics of, 513
malaria and, in British Guiana, 512
prevalence of, in British Guiana, 512
demeilloni, in Mozambique, 112
dureni, malaria and, in Belgian Congo, 210
eiseni
in Salvador, 108
experimentally infected with *P. falciparum*, 515
elutus (*A. sacharovi*)
breeding places of, 429
malaria and
in Greece, 429
Yugoslavia, 430
fluminensis, terminalia of, 513
fluviatilis, 666
in India, 106, 113
malaria and, in India, Nilgiris, 106
funestus
in Mozambique, 112
bionomics, 111
breeding places of, 111
measurement of temperature and light in, 816
control, in East Africa, 671
late seasonal increase in village houses, 358
malaria and
in Belgian Congo, 102, 103, 104, 212, 214, 357
Mozambique, 111
gambiae
in Brazil, 112, 522, 580, 581, 670, 815
Sudan, 288, 665
bionomics, 111
breeding places of, 111, 744
measurement of temperature and light in, 816
control
in Brazil, 670, 815
East Africa, 671
ecology, 580
eggs, viability of, 581
eradication of, from Brazil, 112
malaria and
in Abyssinia, 744
Africa, 631
Belgian Congo, 102, 103, 104, 212, 214, 357
Mozambique, 111
var. *melas*, malaria and, in Africa, 631
gigas
in India, Nilgiris, 106
var. *simlensis*
in India, Nilgiris, 106
hectoris, in Salvador, 108
hispaniola
and *A. sergenti*, differentiation between, (433)
malaria and, in Algeria, 105, 739
hyrcanus
in France, Provence, (667)
India, Udaipur State, 113

Anopheles—cont.
hyrcanus—cont.
in Syria, 110
biology, (667)
morphology, (667)
var. *nigerrimus*, in India, Nilgiris, 106
var. *pseudopictus*
in Italy, 580
biology of, 580
jamesi, in India, Nilgiris, 106
jeyporiensis, in India, 106, 113
karwari, in India, Nilgiris, 106
leesoni, in Mozambique, 112
leucosphyrus, in India, Nilgiris, 106
lindesayi var. *nilgiricus*, in India, Nilgiris, 106
longipalpis, in Mozambique, 112
ludlowi, see *A. sundaicus*
maculatus, in India, Nilgiris, 106
maculipalpis, in Mozambique, 112
maculipennis
Aëdes and, comparative fertility and longevity, 566
control, in U.S.S.R., 519, 520
distribution, in France, (359)
life history, 512
overwintering of, 433
peritrophic membrane in stomach of, 179
phenology of, in U.S.S.R., 287
races
in France, (359)
Germany, 7
geographical distribution of, 7
identification of, 510
race *atroparvus*, 7
malaria and
in Belgium, 214
Portugal, 819
and race *labranchiae*, larvae, differentiation between, (433)
race *labranchiae*, 7
malaria and, in Algeria, 739
and race *atroparvus*, larvae, differentiation between, (433)
race *melanoon*, 7
race *messeae*, 7
breeding places of, 429
engorged, distension of crop of, 434
hibernating, biology of, 434
malaria and
in Greece, 429
Yugoslavia, 430
race *sacharovi*, 7
eggs, pattern of, on surface of water, 287
malaria and
in Albania, 286, 287
Syria, 110
race *subalpinus*, 7
race *typicus*
breeding places of, 429
malaria and
in Greece, 429
Yugoslavia, 430
transport of, in boats, 435
majidi, in India, Nilgiris, 106
marshalli
in Mozambique, 112
var. *moucheti*, malaria and, in Belgian Congo, 214
Anopheles—cont.
minimus
biology of, 436
breeding places of, 666
selection of, 747, 748
control, in Assam, 436
malaria and, in China, 666
ovipositing, relation of shade to, 436
multicolor
eggs, pattern of, on surface of water, 287
malaria and, in Syria, 110
natalensis, in Mozambique, 112
neomaculipalpus, in Salvador, 108
nili, in Mozambique, 112
oswaldoi, characteristics of
eggs, 513
larvae, 513
pallidus
in India, Nilgiris, 106, 112
malaria and, in India, Udaipur State, 112
pharoensis
in Mozambique, 112
Syria, 110
breeding places of, measurement of temperature and light in, 816
malaria and, in Belgian Congo, 357
philippinensis, malaria and, in India, 219, 663
pretoriensis, in Mozambique, 112
pseudopunctipennis
in Grenada, (512)
Salvador, 108
malaria and, in Argentine, 219, 745
punctimacula, in Salvador, 108
punctipennis
flight range of, 748
malaria and, in Guatemala, 292
punctulatus
breeding places of, 816
malaria and, in Melanesia, 816
var. *moluccensis*, in Australia, 870
quadrimaculatus
flight range of, 748, 817
growth, temperature and, 748
malaria and, in U.S.A., 673
oviposition, choice of medium for, 581
resting places of
artificial, 581
diurnal, microclimate of, 667
transmitting *P. lophurae*, 583
ramsayi, in India, Udaipur State, 113
rhodesiensis, in Mozambique, 112
rivulorum, in Mozambique, 112
rufipes, in Mozambique, 112
sergenti
in Turkey, 816
and *A. hispaniola*, differentiation between, 433
malaria and, in Syria, 110
seydeli, in Mozambique, 112
splendidus, in India, Nilgiris, 106
squamosus, in Mozambique, 112
strodei
breeding places of, 515
eggs, characteristics of, 513
subpictus, in India, 106, 113, 114
sundaicus
in India, 9, 219, 663
breeding places, 663

Anopheles—cont.
sundaicus—cont.
habits of, 9
malaria and, in India, 219, 663
superpictus
breeding places of, 429
eggs, pattern of, on surface of water, 287
malaria and
in Albania, 286
Greece, 429
Syria, 110
tessellatus, in India, Nilgiris, 106
theileri, in Mozambique, 112
vagus, in India, 106, 113, 114
varuna
in India, Nilgiris, 106
malaria and, in India, 219
walkeri
biology, 749
breeding places of, 817
characteristics of, 817
feeding habits of, 359
Anopheline Mosquitoes of
Caribbean Region (book review), 808
Northern Half of Western Hemisphere and the Philippine Islands (book review), 954
Anthelmintics
ascaridol, 60, 62, 791
deterioration of, in oil of chenopodium, 922
caprokol, 791
carbon tetrachloride, 62, 615, 925
chenopodium oil, 791
comparative efficacy of, 61
E. 1750, 62
430 Kl., experiments, 60, 61
gentian violet, 619, 932
hexylresorcinol, 60, 615, 791, 925
leche de Higuéron, 612
Lubisan, 62
oleoresin of Aspidium, 323, 925
phenothiazine, 702, 928, 929, 930, 931, 932
value of, 929
santonin, 62, 619, 791, 792, 852
tetrachlorethylene, 62, 325, 615, 619, 925
thymol, 62, 615
Anthrax, in South West Africa, 84
ANTIVENENES AND VENOMS, 81–84, 172–173, 408–410, 485–487, 560–561, 624–626, 861–862, 938–940
Antivenenes
scorpion, 485, 939
preparation of, 82
snake, 264
Aspis aspera, 626
cerastes, 626
Bitis gabonica, 626
lachesis, 626
nasicornis, 626
Bothrops spp., 938
cobra, influence of *p*H on, 862
Dendroaspis viridis, 626
Haemachatus haemachatus, 626
Naja haie, 626
melanoleuca, 626
nigricollis, 81
nivea, 626
spider, *Latrodectus mactans*, 84, 266
Appendicitis
A. lumbricoides and, 556
E. vermicularis and, 328, 402, 555, 927
H. nana and, 556
malaria simulating, 431
N. americanus and, 556
S. stercoralis and, 556
Taenia spp. and, 556
Trichuris trichiura and, 556
Armillifer (*Porocephalus*) *armillatus* infection, in Nigeria, 415
Arthritis, amoebic, 913
Ascariasis
in Algeria, 494
Belgian Congo, 251
Brazil, 579
in children, 550
Bulgaria, 790
Canary Islands, 849
Czechoslovakia, 700
Dutch East Indies, Java, 326
Eire, 852
Germany, 792
Natal, in children, 398
Nigeria, in children, 497
in children, 398, 497, 550, 790, 792, 852
diagnosis by X-ray, 792
treatment (including experimental)
ascaridol, 791
caprokol, 791
chenopodium oil, 791
430 Kl., 60
hexylresorcinol, 791
santonin, 791, 792, 852
Ascaris
eggs, viability of, in pit latrine, 318
lumbricoides
appendicitis and, 556
incidence in U.S.A., Indiana and Kentucky, 148
invasion of lumen of intestine by, 326
obstruction by, in child, 852
pharmacological researches on, 790
suum, experimental eosinophilia with keratin, (554)
Asthma, in Hawaii, 635
Australorbis glabratus
biology of, 849
control of, 849
Avitaminosis, in Nigeria, in children, 497
Babesiasis, treatment by
aromatic diamidines, 377
pharmacological actions of, 682, 683, 684
Bacillus of Vignal-Malassez, healthy rats as carriers of, 776
Bacterium dysenteriae, drying of, 898
Balantidiosis *see* Dysentery, balantidial
Balantidium coli, commensal and parasitic forms, 911
Bartonella bacilliformis
agglutinins for, 137
characteristics of, 770
BARTONELLOSIS, 137–138, 302–303, 769–771, 904–907
in Colombia, 904
Peru, 904
Carrión's disease
in Peru, 905
immunology, 905

Bartonellosis—*cont.*
control, 904
distribution, 770
prophylaxis, 904
transmission, 904
verruga peruviana
in Colombia, 302
diagnosis, 303
immunity, 769
malaria and, 907
phlebitis in, 770
Bejel *see under* **SYPHILIS**
Beriberi
in China, 404
Eritrea, 857
French Equatorial Africa, (168)
Somali troops, 856
diagnosis, differential, from polyneuritis of pseudo-tabetic type, 77
experiments, 795
polished rice deficiencies and, 557
treatment by vitamin B_1, 404
and tropical neuritis, relationship between, 857
vitamin B_1 in relation to, 404
Bessarabia fever *see under* **TYPHUS GROUP OF FEVERS**
Betel chewing, in India, 89
Bila bila
in Bathurst Island, 412
Melville Island, 412
Bilharzia *see* Schistosomiasis
BLACKWATER FEVER and haemoglobinuria, 363–367, 440, 522–524, 584–585, 677–678, 757–758, 826–827, 883–885
in Africa, East, 677
China, Hainan Island, 827
Gold Coast, 585
India, 677
Nigeria, 522
Papua, 884
anuria, 363
blood, lyso-lecithin fragility in, 883
effect of *p*H of blood on haemolysis, 523
haemoglobinuria
in children, following prophylactic quinine, 218
favism and, 722
malaria and, 431, 523, 585
quinine and, 585
serum inhibitor factor in changes in, 584
symptoms, 757
treatment, 523, 585, 758, 827, 885
phenobarbitone, 677
sodium luminal, intramuscularly, 677
Vitex peduncularis, 677
Blastomyces dermatitidis, action of sulphonamides on, *in vitro*, 797
Blastomycosis *see also* Chromoblastomycosis
in Czechoslovakia, 700
Greece, 864
Mexico, 410
U.S.A., 562, 940
complicated by meningitis, 940
epidemiology, 410
symptoms, 410
treatment, 410
X-ray, 562
Blood
of Belgian Congo natives, 262
in bacillary dysentery, chemical analysis of, 393
biotin, influence of, on malaria susceptibility, 825
in blackwater fever, 883, 885
cells, red, mechanism of lysis of, 440
changes, set up by snake venoms, 408
corpuscles, red, formation of, 622
in cutaneous leishmaniasis, 685
Disorders of, Diagnosis, Pathology, Treatment and Technique, 4th edition [WHITBY & BRITTON] (book review), 503
films, staining of, 667
groups, in malaria, 217
haemoglobin levels, in India, Coorg, 170
haemostatic properties of snake venom, (173), 408, 560
in kala azar, 376
leucocytes
enumeration and differentiation of, 860
picture, in typhus, 595
lymphocytes in, of lepers, 787
in malaria-infected monkeys, 584
morphology of, in deficiency anaemia, 329
*p*H, haemolysis and, 523
picture, in typhus, 446
pressure
in typhus, 831
in cerebral arteries, 234
-protein, in tropical pseudotabes, 796
red cell, destruction, mechanism of, 826
sedimentation rate
in Eritrea, 949
in relapsing fever, 950
syphilis, 950
tuberculosis, 950
typhus, 950
BOOK REVIEWS, 96, 182–184, 269–272, 340–344, 415–420, 503–504, 569–570, 641–642, 724–726, 807–808, 873–876, 951–954
Boutonneuse fever *see under* **TYPHUS GROUP OF FEVERS,** tick-borne
Bronchopneumonia in Nigeria, in children, 496
Brucellosis *see* Undulant fever
Bubo, cold inguinal, relationship to plague, 46
Buerger's disease, in Eritrea, 135
Bulinus
resistance to desiccation, 471
contortus raymondi, as intermediate host Schistosoma, 469
Calcaemia, in African natives, (267)
Cancrum oris *see* Noma
Capillaria spp., in cats, in China, 848
Carcinoma of pancreas, and schistosomias, (323)
Cataract
in India, 494
extractions, 943
treatment, 494
Cebus apella, naturally infected with *T. cruzi*, 6
Centrocestus formosanus metacercariae, 254
Ceratitis capitata pupae, soil fumigant destruction of, 501
Ceratopogonidae of Mozambique, (666)

Cerebrospinal fever
in Algeria, 494
British Colonies, 631
Chagas's disease *see* **TRYPANOSOMIASIS,** human, American and *Trypanosoma cruzi*
Child welfare, in Belgian Congo, Mayombe, 177, 178
Chilomastix mesnili infection
in British children, 699
Czechoslovakia, 700
U.S.S.R., 313
Crimea, Sebastopol, 911
U.S.A., Indiana and Kentucky, 148
Chlamydonema praeputiale in cats, in China, 848
CHOLERA, 139–141, 241, 392–393, 454, 539–540, 843–844, 909–911
in Germany, Hamburg epidemic of 1892, (140)
Hong Kong, 843
India, 139, 140, 454
aetiology, 392
control, 843
on airplanes, 564
prophylaxis, 392
Summary of Recent Abstracts, 1–6
transmission, 392
treatment, 392, 843
concentrated saline, 454
non-pyrogenic infusion fluids, 140
sulphonamides (experimental), 843
vaccine, 540
vibrios
involution forms of, produced by glycine, 392
isolation of, 241
lethal action of potassium permanganate on, 910
toxicity of, in animals, 909
V. cholerae
glycolipidic complex in, 910
survival of, in gastric juice, 539
toxicity of, in animals, 910
vomit, examination of, 140
Chromoblastomycosis *see also* Blastomycosis
in Argentine, 491
Cuba, 174
South Africa, 630
aetiology, 174
diagnosis, 631
differential, 174
nasal and laryngeal
in Venezuela, 715
treatment, 715
treatment, 174, 491
Chyladenectasis and steatorrhoea, 483
Cirrhosis
biliary, in Nigeria, in children, 497
hepato-lienal, in Eastern Mediterranean countries, 333
hepatic, in India, 947
Citochol reaction in typhus, 689
Climate
influence of, on disease, 872
physiology and, 872
Clonorchis sinensis infection
in Britain, 157
treatment by collosal antimony, 157
Coccidiosis in Tunisia, 805
Cochliomyia
americana infection in man, (724)
hominivorax, 636
aural infestation with, 340
Colitis
in Nigeria, in children, 497
amoebic, in U.S.A., 697
bacillary, in U.S.A., 697
ulcerative, treatment by sulphaguanidine, sequelae, 242
Compost, use of sullage-water, precipitate for, 640
Conjunctivitis due to
Ficus tumila, 940
lymphogranuloma inguinale, 493
Conus textilis poisoning, 635
Cordylobia anthropophaga larvae, causing cutaneous myiasis, (181)
Corneal vascularization, nutrition and, 620
Crab yaws, in East African campaign, 332
Ctenocephalides felis felis in cats, in China, 847
Culex
fuscanus, susceptibility to experimental infection with *Microfilaria malayi*, 254
pipiens, flight range of, 748
larvae, effects of petroleum oils on, 90
quinquefasciatus, atlas of, 877
Culicines of Tanganyika, 39
Culicoides
respiratory trumpet and anal segment of pupae of, 805
grahami, in Belgian Congo, 214
Culiseta inornatus, flight range of, 748
Cylicospirura spp., in cats, in China, 848
Cysticercosis
cerebral, (850)
in Spain, 65
diagnosis, 65
symptoms, ocular, 65
diagnosis, skin tests, 474
Cysticercus cellulosae infection of brain, 65, (850)
Dacryocystitis
in India, 494
treatment, 494
Dasypus novemcinctus, naturally infected with *T. cruzi*, 526
DEFICIENCY DISEASES, 77–80, 168–170, 259, 404–405, 480–482, 557–560, 619–621, 711–712, 795–796, 854–860, 935–936, *see also under* individual diseases
in India, Mysore, 711
Demodex folliculorum, incidence in skin histology, 872
Demographic studies in Belgian Congo, Nepoko, 495
DENGUE, 604, 694–695, 908–909
Dengue, (604)
in Eritrea, 908
Palau Islands, 870
diagnosis, differential from
Bessarabia fever, 839
leptospirosis, 462
treatment, 909
Depigmentation and oedema syndrome, in Belgian Congo, 406

Dermacentor
andersoni
Rickettsia-like organism from, 238
transmitting Rocky Mountain fever, 534
occidentalis, transmitting Rocky Mountain fever, 534
parumapertus, transmitting Rocky Mountain fever, 534
variabilis
causing tick paralysis, 802
transmitting Rocky Mountain fever, 534
Dermatitis
in Belgian Congo, (176)
Micronesian Islands, 870
due to
Ficus tumila, 940
Grevillea banksii, in Hawaii, 175
necrotic, due to black poplar sawdust, induced by sun's rays, 176
nodular and ulcerative
in Belgian Congo, 148
relationship to yaws, 148
pyrethrum, 723
venenata
caused by *Hippomane mancinella*, 488
Manzanillo tree, in Panama, 863
treatment, 488
Dermatobia
in Colombia, 804
mosquitoes as vectors of, 804
Dermatobia hominis causing myiasis, 93
DERMATOLOGY AND FUNGOUS DISEASES, 173–177, 330–331, 410–411, 487–491, 562, 627–631, 715–716, 797–798, 862–865, 940–941
Dermatoses in West Africa, 945
Desert sores
in East Africa, in troops, 499
sunlight and, 718
treatment, 499
Diamanus montanus and plague transmission, 696
Diamidines, aromatic, pharmacological actions of, 682, 683, 684
Dichlor diphenyl trichlorethane, use in control of lice and other insects, 899
Didelphys virginiana
host of Paragonimus, 65
naturally infected with *T. cruzi*, 526
Dientamoeba fragilis infection, in Czechoslovakia, 700
Diet
survey, in India, Coorg, 170
typhus fever and, 444
Diphtheria in Nigeria, in children, 487
Diphyllobothrium
infection *see under* Tapeworm infection
minus, cycle in development of, 158
spargana, in cats, in China, 848
Diplostomum flexicaudum, development in lenses of mammals, 924
Dipylidium caninum
in cats, in China, 848
infection *see under* Tapeworm infection
Dirofilaria immitis
in cats, in China, 848
larval development, experiments, (709)
Dirofilaria immitis—cont.
transmission, mechanism of
by *Aëdes aegypti*, 71
A. albopictus, 71
Diseases
alimentary, in Nigeria, in children, 497
climate and, 872
communicable from animals, in Australia (499)
deficiency, in China, Shanghai, 404
infectious
of Guam, 716
Japanese Mandated Islands, 716
U.S.S.R., Ukraine, 716
internal, in Fernando Po, 179
Medical, in Tropical and Subtropical Area Memoranda on, 1942 [WAR OFFICE] (book review), 343
non-tropical, hypoprothrombinaemia in, (267)
respiratory
in Africa, West, 945
Bathurst Island, 412
Melville Island, 412
Nigeria, in children, 496
skin, intestinal protozoa and, 797
transmission, rôle of airplane in, 563
tropical
in Australasia. A Handbook, 2nd editio [CILENTO] (book review), 56
hypoprothrombinaemia in, (267)
prevention, (85), (495)
in troops, control, 284
in wartime, (85)
Distomiasis, hepatic, due to *Fasciola hepatic* 472
Dracontiasis
in East African campaign, 332
India, Osmanabad District, 256
Sudan, 633
control, 257
DROPSY, EPIDEMIC, 624
Argemone oil, detection of, 624
Drug
addiction
in India, 87
malaria in, 359
Dysentery, amoebic *see* **AMOEBIASIS**
DYSENTERY, BACILLARY, 51–52, 141–14 241–242, 307–309, 393–39 540–544, 777–781, 844–845
in Abyssinia, 632
Africa
South West, 84
West, in British troops, 385
Australia, 779
Czechoslovakia, 700
Germany, 143
Great Britain, 779
Middle East, in troops, 241, 307, 30 394, 395, 540 ,777
Sudan, 633
U.S.S.R., 143, 459
in German troops, 393
U.S.A., 142, 396, 543, 780
New Orleans, 53
Venezuela, 51
azotaemia in, 393
bacteriology, 543
blood in, chemical analysis of, 393

Dysentery, Bacillary—*cont.*
in children, 396
chloride content of serum in Flexner infections, 51
complications, 542
kidney lesions, 308
control, 543
in Middle East, 540
diagnosis, 394, 396, 541
differential, from
amoebiasis, 541
sandfly fever, 695
sigmoidoscopy, 780
epidemiology, 543
histology, 307
malaria and, 431
morbid anatomy, 141
pathology, 141
sequelae, disturbed function of small intestine, treatment, 143
symptoms, 394
treatment
bacteriophage, 394
and sulphonamides, comparative value of, 845
kaolin, 394
methylene blue, 143
sodium sulphate, 394, 780
sulphonamides, 241, 777, 779
and phage, comparative value of, 845
succinyl sulphathiazole, 143, 396, 544, 780
sulphaguanidine, 54, 142, 308, 394, 395, 542, 543, 777, 779, 780, 781
complications, 780, 781
sulphapyridine, 844
haematuria following, 242
in troops, 52, 385, 241, 307, 308, 385, 393, 394, 395, 540, 777
Dysentery bacilli
in Venezuela, 51
isolation of, 541
Dysentery
balantidial, (313)
in Mexico, 460, (782)
U.S.S.R., 313, 459
Crimea, Sebastopol, 911
in children, 459
control, in closed institutions, 460
giardial
in Poland, 147
diagnosis, 147
treatment by atebrin, 54
Ear infestation of, by *Cochliomyia hominivorax*, 340
Echidnophaga gallinacea and plague transmission, 696
Echinococcosis *see* Hydatid disease
Echinostoma ilocanum, invasion of lumen of intestine by, 326
Echinostomiasis, in Java, 326
Eclampsia, malarial, 289
Elephantiasis *see* Filarial lymphangitis
Encephalitis
diagnosis, differential, from trench fever, 839
meningo-, trypanosomal
diagnosis, 120
treatment, 120
in relapsing fever, 461
Encephalitis—*cont.*
trichiniasis and, (328)
yellow fever, 44
immunity experiments, 772
Encephalomyelitis, equine, virus
action of vapour of eucalyptus on, 198
and rabies virus, para-immunity phenomenon between, 661
Endarteritis obliterans, juvenile *see* Buerger's disease
Endocarditis, bacterial, leprosy and, 464
Endolimax nana
incidence in Moscow sewage, 311
infection
in Czechoslovakia, 700
U.S.S.R., 313
Crimea, Sebastopol, 911
U.S.A., Indiana and Kentucky, 148
Entamoeba
coli
in U.S.S.R., 313
Moscow sewage, 311
commensal and parasitic forms, 911
cysts, fluctuation in numbers of, in stools of monkeys, 456
infection
in British children, 699
U.S.A., Indiana and Kentucky, 148
gingivalis
cultivation of, 147
and *E. histolytica*, differentiation, 147
infection, in Mexico, 455
histolytica
in Crimea, Sebastopol, 911
Moscow sewage, 311
U.S.A., Indiana and Kentucky, 148
causing penile ulcer, 456
commensal and parasitic forms, 911
cysts
of duodenum, 606, 781
fluctuation in numbers of, in stools of monkeys, 456
viability, effect of chlorination of water on, 782
encystment
in culture, effect of *p*H on, 310
and maintenance of pathogenicity of, relationship between, 455
and *E. gingivalis*, differentiation, 147
growth, in culture, effect of sulphonamides on, 455
house-fly as carrier of, 698
races of, 53
varying virulence of strains of, 310
Enterobius vermicularis infection
in Algeria, 494
Australia, 932
Brazil, in children, 550
Bulgaria, 790
Canada, 617, 710
in children, 328
Canary Islands, 849
Czechoslovakia, 700
Mexico, 617
Philippine Islands, 617
U.S.A., 148, 617, 618, 927
in children, 710

Enterobius vermicularis infection—*cont.*
in appendix, 328, 402, 555, 927
in children, 328, 550, 710, 790
diagnosis
Graham swab, 617
NIH swab, 617, 618, 710
symptoms, 929
treatment, 63
ascaridol, experiments, 60
gentian violet, 619, 932
phenothiazine, 928, 929, 930, 931, 932
toxicity of, 928, 929, 930, 931, 932
santonin, 619
tetrachlorethylene, 619
of wall of Fallopian tube, 403
Entomobrya multifasciata, 805
tenuicauda, 805
Entomology in Mozambique, (180)
Entropion
trachoma and, 942
treatment, 942
Eosinophile-lung in India, 720
Eosinophilia, tropical
in India, 407
treatment, 407
Epididymofuniculitis, filarial
in Cape Verde Island, 709
Martinique, 709
Epilepsy, malaria and, 431
Espundia *see* **LEISHMANIASIS,** muco-cutaneous
Eurytrema spp. in cats, in China, 848
Eutriatoma
oswaldoi, naturally infected with *T. cruzi*, 525
sordida, naturally infected with *T. cruzi*, 525
Eye
diseases, of Eritrea, (494)
infestation by nematode, 554
injury to, by sun, 494
lesions, due to *Naja nigricollis* venom, 81
Fasciola hepatica
in cats, in China, 847
causing hepatic distomiasis, 472
developmental cycle of, (65)
infection, cutaneous and intracutaneous tests in, 253
Favism
in France, 722
Palestine, (722)
haemoglobinuria, 722
Felis domestica, parasites of, in China, Kweiyang, 847
Fever, ten-day, simulating typhoid fever, in Ceylon, 239
Ficus tumila causing
conjunctivitis, 940
dermatitis, 940
Filaria conjunctivae
in Italy, 927
in premammary region, 927
Filarial lymphangitis
in Assam, 160
Gabon, 72
scrotal, surgery of, 72
Filariasis, *see also* Wuchereria, Loa, Onchocerca, Dracunculus, Dirofilaria
in Africa, East, 616
campaign, 332
Assam, 160
Cameroons, 160
Filariasis—*cont.*
in Palestine, in negro troops, 926
leprosy and, 149
Fish
nicotinic acid content of, 80
poisonous of Porto Rico, 86
Fleas, (267)
of U.S.A., 775, 841
Flies, (267)
Fly
control, in Egypt, 91
house-
as carrier of intestinal protozoa, 698
life history 572
population, of China, 871
sprays, pyrethrum activation, 338
Fonsecaea spp., behaviour of, 410
Food
pests, identification of, 950
Primer for Use in Schools, Colleges, Welfare Centres, etc., in Afric [McCARRISON *et al.*] (book review), 953
Fungicides and Insecticides, Chemistry of (book review), 807
Gale filarienne in loa loa infection, 852
General paralysis of the insane and experimental relapsing fever, 315
Gentian violet in Enterobius infection, 619, 932
German measles, diagnosis, differential, from typhus, 299
Giardia infection
in Porto Rico, 459
in children, 313, 459, 699
treatment by atebrin, 459
Giardia
intestinalis
in U.S.S.R., 313
Moscow sewage, 311
infection
in British children, 699
Czechoslovakia, 700
U.S.S.R.
in children, 313
Crimea, Sebastopol, 911
U.S.A., Indiana and Kentucky, 148
symptoms, 700
treatment
acranil, 700
atebrin, 699, 700
mepacrine, *see* atebrin
quinacrin, 700.
lamblia see *Giardia intestinalis*
Glaucoma
in India, 494
treatment, 494
Glossina
of Kenya, 293, 294
bionomics of, 524
control
in British Colonies, 631
Kenya, 678
Rhodesia, 588
trapping, 885
dissection of, for detection of trypanosomes, 1
distribution of, in Mozambique, 524
habits of, 293, 294
life history, 512

Glossina
austeni, transmitting *T. congolense*, 887
brevipalpis, polymorphic trypanosome infections found in salivary glands of, 369
palpalis, transmission of *T. gambiense* by, irregularities in, 368
Goitre
in French Equatorial Africa, 948
treatment, 949
Granuloma
coccidioidal, in U.S.A., 411
venereum, of eyelid
in U.S.A., 493
treatment by tartar emetic, 493
Granulomata, amoebic, causing intestinal obstruction, treatment, 144
Grevilea banksii causing dermatitis, in Hawaii, 175
Guinea worm *see* Dracontiasis *and* Dracunculus
Habaswein itch in Kenya, 562
Haemogogus spp.
in Salvador, 108
relation to jungle yellow fever, 537
Haemophysalis bispinosa
experimentally infected with Q fever, 601
leporis palustris
seasonal history of, 836
transmitting Rocky Mountain fever, 534
HAEMATOLOGY, 170–172, 262–263, 329, 405–408, 484–485, 622–624, 714–715, 860–861, 938
Haematuria, paroxysmal, due to malaria, 668
Haemoglobinuria *see under* **BLACKWATER FEVER**
Harara
in Anatolia, 339
Syria, 339
sandfly fever and, in Palestine, 338, 339
Health
of Sandawe Tribe, Kondoa Irangi, Tanganyika, 867
Department of, Brazil, report for 1941 (book review), 182
and fitness, in tropics, 93
Handbook of, for Overseas Service [SHATTUCK & MIXTER] (book review), 953
public, of West Indies, 632
services
in Morocco in 1941, (563)
South-West Africa, since 1918, 84
in tropics, 806
Heat
collapse, treatment, 94
cramps, treatment, 94
exhaustion, treatment, 719
and moisture, dissipated from body by respiration, calculation of, 502
stroke, 268
sequelae, 94
treatment, 94
Heights and weights of Cantonese adult males, (96)
Helminths
appendicitis and, 328, 402, 555, 556, 927
in cats, in China, 848
eggs
dilution counts of, natural pattern of, 549
risk of contaminating vegetables manured with sewage, 318
Helminths—*cont.*
eggs—*cont.*
viability of, in pit latrines, 318
parasites, faeces examination, sedimentation technique, 550
HELMINTHIASIS, 60–77, 155–168, 249–258, 318–329, 398–403, 466–480, 549–556, 612–619, 702–711, 790–795, 847–854, 922–935, *see also under* names of worms
in Africa
South-West, 84
West, 702
Australia, (923)
Brazil, Minas Geraes school children, (63)
Canary Islands, 849
Summary of Recent Abstracts, 571–578, 643–650
treatment
430 Kl., experiments, 60, 61
phenothiazine, 702
Hemeralopia in Eritrea, 856
Herpes
febrilis, in relapsing fever, 461
virus, association of, with rabies street virus, 206
Hippelates, in U.S.A., 339
Hippomane mancinella causing dermatitis venenata, 488
Histoitus velatus, susceptibility to typhus fever, (136)
Histoplasmosis
in South Africa, 176
U.S.A., 177, 330, 798
experimental, 331
of knee, in U.S.A., 177
with mucocutaneous manifestations, 330
producing vegetative endocarditis, 798
treatment
neostam, 330
sulphathiazole, 330
tartar emetic, failure of, 798
Hookworms
Ancylostoma
brasiliense
in China, in cats, 848
Dutch East Indies, Java, 326
invasion of intestinal wall by, 326
caninum, in cats, in China, 848
duodenale, invasion of intestinal wall by, 326
disease *see* Ankylostomiasis
Necator americanus
appendicitis and, 556
incidence, in U.S.A., Indiana and Kentucky, 148
infection *see under* Ankylostomiasis
invasion of lumen of intestine by, 326
ova
counting of, 67
preservation of, 707
Hoplopsyllus anomalus, and plague transmission, 696
Hospital construction, in tropics, 95
Housing, tropical, 95, 638
Hydatid disease, (926)
in Wales, 850
diagnosis, 851

Hydatid disease—*cont.*
hypersensitivity in, 474
of kidney, (66)
of lung, (66), (795), (926)
diagnosis, 614
treatment, (254)
symptoms, 851
of thyroid, (66)
treatment, 851
of uterus, (614)
Hymenolepididae, cestodes of, (400)
Hymenolepis
diminuta,
infection *see under* Tapeworm infection
nutrition of, 706
nana
appendicitis and, 556
incidence, in U.S.A., Indiana and Kentucky, 148
infection *see under* Tapeworm infection
var. *fraterna*
infection *see under* Tapeworm infection
serology of, 851
Hyperkeratosis palmaris et plantaris, ainhum and, 85
Hypoglycaemia, in trichiniasis, (619)
Infant
mortality
of Algeria, 494
Ceylon, 854
Nigeria, 496
South Africa, Transkei, 865
Infection, resistance to, temperature and, 567
Influenza
in South-West Africa, 84
diagnosis, differential, from
sandfly fever, 695
trench fever, 839
Insecticides
diphenyl trichlorethane, 899
and Fungicides, Chemistry of (book review), 807
pyrethrum, dermatitis, 723
Insects of Micronesian Islands, 870
Intestinal obstruction by amoebic granulomata, treatment, 144
Iodamoeba butschlii
in U.S.S.R., 313
Moscow sewage, 311
U.S.A., Indiana and Kentucky, 148
infection
in British children, 699
Crimea, Sebastopol, 911
Czechoslovakia, 700
Ixodes holocyclus transmitting Q fever, 388
Japanese river fever *see* **TYPHUS GROUP OF FEVERS,** tsutsugamushi disease
Jaundice
following yellow fever inoculation, 40, 41
haemolytic, familial and sickle-cell anaemia, similarity between, 714
Jersild syndrome
in Cameroons, 803
aetiology, 803
symptoms, 803
treatment, 804
Kahn
reaction in typhus, 689
verification test, in malaria, 820
Kala azar *see under* **LEISHMANIASIS**
Keratitis
parenchymatous, following riboflavin deficiency, 936
superficial
in India, Madras, 491
following riboflavin deficiency, 491
treatment, 491, 492
Keratoconjunctivitis
in India, 492, 494
U.S.A., 493
and adenitis,
in India, Calcutta, 492
treatment by sulphanilamide, 492
Keratomalacia
in Ceylon, 854
India, 494
Kidney lesions in bacillary dysentery, 308
Labour
conditions
of Ceylon, 866
Malaya, 866
Mauritius, 866
supervision, in British Colonial Empire, 1937–1943, (866)
Larvicides *and see under* Anopheles mosquitoes
calcium arsenite, 742, 753
copper arsenite, 17
cotton-seed tar, 672
distillate from coke stills, 521
freon-pyrethrum aerosol, 882
mineral oils, comparative suitability of, 16
Paris green, 219, 672, 673, 754
-lime mixtures, 16
petroleum, 219
Lathyrism
in Tanganyika, 869
aetiology, 87
transmission, experiments, 87
Latrodectus
curacaoensis in Colombia, 486
mactans
in Chile, 83
U.S.A., 83
bite of, 83, 939
treatment, 266, 862
Leech infection, in Abyssinia, in child, 869
Leishmania
brasiliensis
experiments with, 230
isolation of, 442
tropica, rôle of methaemoglobin on leptomonad phase of, 378
LEISHMANIASIS, 23–35, 120–125, 227–230, 295–297, 375–378, 441–442, 527–528, 590, 682–685, 765, 828, 891–892
in Brazil, 546
São Paulo, 266
Venezuela, 25
Ashkhabad sore, 296
canine, in Bulgaria, 121
control, in Brazil, 546
cutaneous *see also* oriental sore
in British Guiana, 442
Colombia, 442
Kenya, 230
Mexico, Usumacinta Valley, 453
U.S.S.R., 24

Leishmaniasis—*cont.*
cutaneous—*cont.*
blood in, 685
(gum collectors' ulcer), in Mexico, 441
(Pendeh sore), 296
in U.S.S.R., 441
treatment, blood-dressings, 441
relapses, 25
treatment
neostibosan, 25
tartar emetic, 25, 442
dermal
in India, 121
complications, 121
treatment by urea stibamine, 121
gum collectors' ulcer *see under* cutaneous
induced, in paretics, 685
kala azar
in Abyssinia, 229, 632
Algeria, in children, 892
Argentine, 892
Bolivia, 892
Brazil, 892
China, 229
East African campaign, 332
India, 121
Mediterranean countries, 376
Spain, 23, 120, 375
Sudan, 23, 122, 684
U.S.S.R., 295
Venezuela, 891
blood in, 376
bone marrow changes in, 376
Chemotherapy [BRAHMACHARI] (book review), 417
in children, 23, 295, 892
diagnosis, 121, 892
complement fixation test, 762
spleen puncture, 376
sternal puncture, 376
infantile, 23
in Algeria, 892
Spain 23.
transmission, (23)
experiments, 227
treatment
aromatic diamidines, 23, 121, 122, 684
pharmacological actions of, 682, 683, 684
neostibosan, 120, 892
and quinine, 892
sodium salt of mannite and antimonic acid, experimental, 229
solustibosan, 229
stilbamidine, 121
fluorescence and adsorption of, 122
reactions following, 122,
toxicity of old solutions, 23
of lymph glands, in China, Peiping, 228
muco-cutaneous (espundia)
in Argentine, 125
treatment by tartar emetic, intravenously, 125
oriental sore
in Algeria, 494, 527, 590
in child, 528
Eritrea, 124
Turkey, 125
U.S.S.R., 296, 297
U.S.A., 377

Leishmaniasis—*cont.*
oriental sore—*cont.*
forms of, in U.S.S.R., 296
treatment, 125, (378)
antimonials, 828
atebrin, 124, 125, 527
quinacrine, *see* atebrin
tartar emetic, 377
vaccine, 297
X-ray, 25
Pendeh sore, *see under* cutaneous
Summary of Recent Abstracts, 421–427
treatment
aromatic diamidines, experiments, 377
neostibosan, 228
stilbamidine, increased toxicity of, on exposure to light, 376
LEPROSY, 57–60, 149–155, 245–249, 315–317, 397–398, 462–465, 546–549, 609–611, 700–702, 784–790, 847, 921–922
in Africa, South-West, 84
Antigua, 463
Balkans, 59
Barbados, 463
Bathurst Island, 412
Belgian Congo, 316, 397
Brazil, Maranhão, 150, 921
Bulgaria, 59
Burma, 153
Colombia, 248, 549
Cook Islands, 315
Crete, 59
Dutch East Indies, 152
Fiji, 315, 700
French Equatorial Africa, 155
Gilbert Islands, 315
Greece, 59
India, 57, 397, 546, 784, 785
Jamaica, 462
Kenya, 154
Lithuania, 245
Malaya, 153
Maltese troops, (248)
Melville Island, 412
Mexico, 609
Montenegro, 59
Philippine Islands, Culion, 464, 465
Portugal, 60
Rumania, 59
St. Kitts and Nevis, 463
St. Lucia, 463
South Africa, 611
Spain, 60
Spanish West Africa, 149
Sudan, (609), 633
Trinidad, 463
U.S.A., 609, 610, 847
Yugoslavia, 59
adenopathy in, 547
aetiology, 785
bacterial endocarditis and, 464
blood lymphocytes in, 787
British Empire Leprosy Relief Association annual report 1941, 57
(Madras), annual report 1941–42, 397
in children, 397
of leprous parents, 149
control, 317

Leprosy—*cont.*
classification of, 786
colocasia and, 701
complications
filariasis, 149
helminthiasis, 149
malaria, 149
trypanosomiasis, 149
control, 785
in Brazil, 150
Fiji, Makogai Leper Hospital, annual report 1940 and 1941, 315, 700
India, 397, 784, 785
Jamaica, Spanish Town asylum, 462
Spanish West Africa, 149
Trinidad, 463
U.S.A., Carville Leprosarium, 847
segregation, 58
in tropical rural areas, 155
diagnosis
lepromin test, 245, 397, 548
sweating dysfunction, 397
duration, sixty-one years, (248)
epidemiology, 785
erysipelatoid manifestation, treatment by sulphanilamide, 609
histology, 787
injection set, portable, (317)
inoculation into hamsters, 150
lepra reaction, 787
treatment by fouadin, intramuscularly, 785
lepromin test, 152, 245, 316, 317, 397, 548, 786, 787
lesions, 785
Lucio form, histopathology of skin in, 922
Makogai Central Leper Hospital, annual reports 1940 and 1941, 315, 700
Mitsuda test *see* lepromin test
neural, 149, 315
in Fiji, 700
treatment
caloncoba oil, 316
Dilester, 316
ganglion infiltration in, 155
vitamin B_1, failure of, 245
nodular, treatment, (789)
pathology, 151, 785
prognosis, 151
rat leprosy
in Brazil, (922)
pathology, (922)
Research Department, Calcutta School of Tropical Medicine, report 1941, (546)
roseola and, 788
Summary of Recent Abstracts, 809–815
transmission, 785
hereditary, 149
insects, 151
treatment, 149, 151, 316, 785
caloncoba oil, 316
chaulmoogrates, 316, 549
chaulmoogra oil, 245, 465
and benzocaine, 609
intramuscularly, 701
chemotherapy, 610

Leprosy—*cont.*
treatment—*cont.*
diphtheria antitoxin, 154
ethyl esters, 701
lactoflavine, 702
N.A.C., 611
serum, tests, 152
Stibicol, 248
sulphanilamide, 610
experimental, 789
sulphathiazole, 789
X-rays, harm of, 245
tropho-neural, ? cure of, 789
tuberculoid
in India, 701
Nigeria, 701
U.S.S.R., 701
? cure of, 789
lesions, seasonal variations in activity of, 701
para-arterial neural infiltration, 547
tuberculosis and, 847
ulcers, treatment, closed plaster, 702
Leptospira
icterohaemorrhagiae and *L. pomona*, differentiation between, 918
mitis n. sp., 918
pomona n. sp., 918
and *L. icterohaemorrhagiae*, differentiation between, 918
Leptospirosis
in Abyssinia, 633
Africa, 462
India, 783
U.S.A., 304, 919
diagnosis, differential from
dengue, 462
relapsing fever, 462
yellow fever, 462
meningitis and, 919
yellow fever and, pathological differences between, 304
Leukomata, corneal, in India, Calcutta, 492
Lice, (267)
control, 832
diphenyl trichloroethane, 899
fumigants, toxicity of, 601
lethane body belt, 900
Liver abscess *see under* **AMOEBIASIS**
Loa
extraocularis infection, in U.S.S.R., 326
loa
in Belgian Congo, 214
Cameroons, in American missionaries, 926
Gabon, 852
Sierra Leone, 852
diagnosis, 852
treatment
antimony and potassium tartrate, failure of, 927
mapharsen, 927
Loeffler's syndrome, 400
in Venezuela, 721
aetiology, 721
Louping ill virus, association of, with rabies street virus, 206
Lymphangitis, streptococcic, diagnosis, differential from sporotrichosis, 490

Lymphogranuloma
inguinale, complications, ocular, 493
venereum, treatment, experimental, by sulphonamides, effect of para-amino-benzoic acid on, 756
Madras Government Ophthalmic Hospital, annual report 1941, 494
MALARIA, 6–17, 101–119, 210–223, 283–293, 357–363, 427–440, 510–522, 579–584, 663–677, 738–757, 815–826, 877–883
in Abyssinia, 632, 686, 744
Africa, 631
South-West, 84
West, in British troops, 385
Albania, 286, 287
Algeria, 105, 430, 494, 738, 739, 740, 741
Angola, 744
Argentine, 219, 745
Belgian Congo, 102, 103, 104, 210, 211, 212, 213, 218, 357, 743
Belgium, 214
Brazil, 510, 514, 515, 518, 546, 579, 670, 746, 824
São Paulo, 266, 513
British Guiana, 512
Ceylon, 746
China, 666
Cuba, 431, 745
Czechoslovakia, 217
Dutch East Indies, 107, 750
East African campaign, 332
Eritrea, 880, 950
Germany, (108), 669
Great Britain, 518, 579
Greece, 292, 427
Guatemala, 292
India, 219, 663, 668, 673, 749, 753
Coorg, 170
Nilgiris, 105
South, 116
Udaipur State, 112
Lebanon, 110
Melanesia, 816
Mexico, Usumacinta Valley, 453
Morocco, 742
Mozambique, 110
Nigeria, in children, 497
Pacific Islands, 107
Philippine Islands, 13
Porto Rico, 753
Portugal, 818
Epidemiology of [CAMBOURNAC] (book review), 951
Salvador, 107, 267
Spain, 751
Sudan, 633
Syria, 110
Tanganyika, 879
U.S.S.R., 431, 433, 436
in children, 436, 668
U.S.A., 109, 215, 290, 436, 579, 664, 665, 673, 819
in navy, 822
troops, 283
Yugoslavia, 429
agriculture and, 753
ankylostomiasis and, anaemia due to, 431
Malaria—*cont.*
atypical, 6, 819, 820
avian, 286
attempted infection of man with, 756
in ducks, 439
susceptibility of, to, 683
treatment by sulphonamide, 223
in ducklings, treatment by sulphonamides, effect of para-aminobenzoic acid on, 756
fatal anaemia in, 293
fowl, 116, 117
immunization, 362
influence of biotin on susceptibility to, 676
intravascular agglutinations in, 676
treatment
acranil, 439
chemotherapy, 17, 223
comparative, 879
in turkeys, in Kenya, 363
blackwater fever and, 431, 523, 585
blood
groups in, 217
sedimentation rate, in Eritrea, 950
cardiac lesions in, 10
cerebral, 431
in children, 102, 103, 107, 218, 428, 436, 497, 668, 744, 818.
central nervous system in, 517
complications, 516
fatal case of *P. falciparum* infection, 436
treatment, quinine tannate, 291
complications, 516, 579
paroxysmal haematuria, 668
control, 882 *and see* prophylaxis
in Africa, West, 944
Algeria, 105, 430, 740
Argentine, 219
Cuba, 431
Greece, 429
Guatemala, 292
India, 219, 673, 752
Morocco, 742, 753
Porto Rico, among troops, 753
U.S.A., 579, 673
Tennessee, 15
U.S.S.R., (14)
Yugoslavia, 430
public health aspect, 752
diagnosis, 754
blood films
staining of, 667
thick, 743, 750
complement fixation, 11
differential, from
sandfly fever, 695
trench fever, 839
flocculation test, 290, 360
microscopical, 432
distribution, 284
in drug addicts, 359
dysenteric, 431
eclampsia and, 289
epileptiform, 431
experimental, complement fixation reaction in, 118
falciparum, treatment, quinine, resistance to, 880
immunity, in U.S.S.R., 431

Malaria—*cont.*
in infant, 6
Kahn verification test in, 820
kidney in, 360
leprosy and, 149
masked
in Croatia, 291
diagnosis, 291
treatment, 291
plasmoquine and quinine, 291
meningococcal septicaemia simulating, 216, 217
in monkeys
blood in, 584
pathology of, 118
morbidity, and parasitaemia, relationship between, 819
in navy, 215
nephritis and, 360
pernicious, treatment, 878
P. falciparum infection, 9
pathological findings, 436
P. vivax infection, complement fixation of serum with *P. knowlesi* antigen, 750
pregnancy and, 289
termination of, 431
premunition of African natives against Algerian strain, 741
problem, in overseas troops, 877
prophylaxis, 882
atebrin, 821, 822, 944
drugs, economy in, 750
iodo-mercurate of manganese, 824
quinine, 741, 742, 751, 944
haemoglobinuria following, 218
Recent work in Russia (special article), 345–351
relapses, 13
treatment, 751
rheumatic, 431
rice fields and, (877)
serum reactions, 669
service, of Morocco, annual report 1939, (284)
simulated, in Belgian Congo, Ruanda, 212
simulating appendicitis, 431
spleen, spontaneous rupture of, 218, 820
splenomegaly, treatment by calcium chloride, 13
Summary of Recent Abstracts, 185–195, 273–283
susceptibility, influence of biotin on, 825
symptoms, 749
tertiana perniciosa siderans, in U.S.S.R., 668
therapeutic, 285
in U.S.A., Florida, 11
liver function in, 582
plasma protein in, 10
treatment
interruption of, 751
thio-bismol, 752
selective action of, 881
transmission *see* Anopheles
index of, 101
treatment, 285, 518, 750, 754, 821, 879
Alstonia scholaris, failure of, 670
[ALVARADO] (book review), 272
atebrin, 9, 291, 437, 820, 821, 822, 823
morphological changes in *P. falciparum* following, 292

Malaria—*cont.*
treatment—*cont.*
atebrin—*cont.*
sequelae, mental, 582
toxicity of, 751
atebrin and plasmoquine, 13, 519, 585
relapse following, 13
versus quinine, 822
atebrin and quinine, 518, 822
atebrin and sulphadiazine, 823
atebrin musonate, 823
Atepé, psychosis following, 751
certuna, 292
chemotherapy, advances in, 179
drugs, economy in, 750
gamefar, 218
iodo-mercurate of manganese, 824
mepacrine *see* atebrin
neosalvarsan and quinine, 291
plasmoquine, toxicity of, 751
plasmoquine and atebrin, 13, 519, 585
plasmoquine and quinine, 291
quinarsol, 360
quinine, 13, 290, 360, 431, 437, 518, 585, 668, 751, (821)
agranulocytosis following, (821)
diuresis following, 360
toxic amaurosis, due to, 290
versus atebrin, 822
quinine and atebrin, 518, 822
quinine and neosalvarsan, 291
quinine and plasmoquine, 291
quinoplasmoquine, 669
relapses, 670
sulphadiazine and atebrin, 823
totaquine
composition of, 879
standards for, 290
Vitex peduncularis, 677
in troops, control, 283, 284, 753
typhus and, 686
urine in, 113
urticaria and, 359
verruga peruviana and, 907
vitamin C and, 13
vivax, immunity, 818
wartime, (9)
Wassermann reaction and, 669
Malnutrition, in South Africa, Transkei, 865
Mansonella ozzardi
in Argentine, 476
Colombia, 476
Dominica, 476
Dutch Guiana, 476
Mexico, 476
Panama, 476
St. Lucia, 476
Mansonoides in Mozambique, 112
Manzanillo tree causing dermatitis venenata, 863
Measles
in Algeria, 494
Sudan, 633
Media, liquid, aeration of, 390, 391
Medical
conditions
in Bathurst Island, 412
Melville Island, 412

Medical—*cont.*
Diseases, in Tropical and Subtropical Areas, Memoranda on, 1942 [WAR OFFICE] (book review), 343
Parasitology [CULBERTSON] (book review), 873
problems
in British Colonies, in wartime, 631
of the Transkei, South Africa, 865
service, for West African air lines, 943
Tropical, Manual. For use and Guidance of Salvation Army Officers on Missionary Service, 3rd edition [TURNER] (book review), 875
Medicine, Tropical
American, Story of, Ambassadors in White [WILSON] (book review), 417
synopsis of [MANSON-BAHR] (book review), 415
Medusa poisoning, treatment, 722
Megalopyge opercularis poisoning, 181
Meinicke reaction, in typhus, 689
Melanosis of Riehl, in Mexico, 482
Melioidosis
in Indo-China, 85
chronic, in Britain, 498
Pf. whitmori isolated in, 85
transmission, 85
treatment, 498
Meningitis
in Nigeria, in children, 497
cerebrospinal, in South-West Africa, 84
complicating blastomycosis, 940
epidemic, diagnosis, differential from typhus, 299
leptospirosis and, 919
Meningococcal septicaemia, simulating malaria, 216, 217
Mesocestoides variabilis, hosts of, 324
Microfilaria
loa, duration of life of, 161
malayi, susceptibility of *Culex fuscanus* to experimental infection with, 254
perstans, duration of life of, 161
Microtus mexicanus, experimentally infected with *S. turicatae*, 314
MISCELLANEOUS, 84–96, 177–182, 266–269, 331–340, 412–415, 494–503, 563–569, 631–641, 716–724, 799–807, 865–873, 943–951
Molluscs of Belgian Congo, 249
Monopsyllus anisus, in cats in China, 847
mortality
child, in Belgian Congo, 177, 178
infant
in Algeria, 494
Ceylon, 854
Nigeria, 496
South Africa, Transkei, 865
Mosquitoes
of Assam, 160
British, control of, 413
Guiana, (870)
Pacific, (9)
Salvador, 108
Abatement, Alameda County, annual report 1942, (636)
Mosquitoes—*cont.*
Anopheline
of Caribbean Region (book review), 808
of Northern Half of Western Hemisphere and the Philippine Islands (book review), 954
control, 413, 670, 671
airplane in, 17
in airplanes, 564, 908
alkaloidal fogs, 15
automatic siphon, 674
disinsectization of day time shelters, 521
ditch lining, 12, 15, 673
drainage, 219, 673
fish breeding, 114, 430, 431, 437, 494, 673, 741
flushing, (825)
irrigation, 673
of ricefields, 114
laboratory findings, 413
larvicides, 335
calcium arsenite, 742, 753
copper arsenite, 17
cotton-seed tar, 672
crushing strength of biological films on natural waters and, 582
distillate from coke stills, 521
freon-pyrethrum aerosol, 882
mineral oils, comparative suitability of 16
Paris green, 219, 672, 673, 754
automatic distribution, 115
-lime mixtures, 16
petroleum, 219
use of waste lubricating oil in, 293
oiling, 754
pyrethrum extract, 14
repellants for, 179, 180
spraying, 116, 882
sullage on ricefields, 112
Xenopus clivii, 744
flight range of, 748
found in rot-holes in trees and bamboo stumps, in Ceylon, 804
larvae, living, transportation, 334
male terminalia, staining, dissecting and mounting of, 334
Plasmodium gallinaceum transmission to, 362
Studies, in U.S. military establishments, (413)
tank-breeding, control of, in Portsmouth, 870
toxicity of aerosols from spraying solutions of insecticides in liquefied gas, 337
Mus musculus, naturally infected with *T. cruzi* 526
Musca
domestica
larvae, influence of temperature on, 499, 500
pupae, soil fumigant for destruction of, 501
larvipara, life history, 512
vicina, biology of, (93)
Mycetona
in Cuba, 630
Mexico, 489
due to *Actinomyces mexicanus*, 489
symptoms, 489

Mycobacterium
leprae, viability after sulphanilamide treatment, 790
pseudotuberculosis and *Past. pestis*, differentiation of, 139
Mycoses, surgical, in Greece, 864
Myelitis, acute, following antirabies vaccination, 204
Myiasis
in U.S.S.R., 871
conjunctival, due to *Oestrus ovis*, (340)
cutaneous, due to *Cordylobia anthropophaga* larvae, (181)
due to *Dermatobia hominis*, 93
ocular
in Algeria, (566)
Egypt, (566)
due to *Oestrus ovis*, (566)
of tongue, in Bulgaria, 93
treatment, surgical, 93
of vulva, (567)
Myositis tropica
in Cameroons, 719
Gold Coast, 719
Nigeria, 719
Necator *see under* Hookworms
infection *see under* Ankylostomiasis
Nematode, infesting eye, in South Africa, 554
Neotoma micropus micropus, naturally infected with *T. cruzi*, 526
Nephritis, malarial, 360
Neurasthenia, tropical, 634
Neuritis
peripheral, diagnosis, differential, from tick paralysis, 802
tropical
in Eritrea, 857
and beriberi, relationship between, 857
heart-block in, 857
treatment, 857
and tropical pseudo-tabes, differential diagnosis, 857
typhus and, 899
Neuropsychiatry, tropical, 802
Niacin deficiency in disease, 169, 559
Nippostrongylus muris
immunization of rats against, 792
infection
experimental, relation of vitamin A to, 793
histopathology of skin, lungs and intestine of rats during passive immunity to, 319
in rats, passive immunity, 319
Nocardia madurae, morphology, 940
Noma
in India, 947
treatment by urea stibamine, 947
Nursing, Tropical. A Handbook for Nurses and others going abroad [GREGG] (book review), 641
Nutrition
in British Colonies, 631
Ceylon, 854
Nigeria, 480
Nutritional degeneration, and tryparsamide amblyopia, 679
Oedema, nutritional
in Salvador, in children, 712
South Africa, Transkei, 865
Oedema, nutritional—*cont.*
in children, treatment, 712
Oestrus ovis causing ocular myiasis, (340), (56
Onchocerca spp.
life history of, 164
volvulus
development in Simuliidae, 74
inflammatory reaction of nodules of, 327
transmission by Simuliidae, 162
Onchocerciasis
in Belgian Congo, 162, 167, 214, 327
East Africa, in West African natives,
Gold Coast, 73
Kenya, 73
Senegal, 848
U.S.A., 709
diagnosis, 73
differential, 167
microfilariae
detection of, in eye, 493
in vitreous, ophthalmoscopic observation o
709
ocular, 616
problem of, on Pan-American highway, 255
symptoms, 165
treatment
Bayer 205, 74
euflavine (Gonacrin), 74
tryparsamide, 74
OPHTHALMOLOGY, TROPICAL, 491–49
941–943
Opisthorchiasis in U.S.S.R., 613
Opisthorchis felineus, intermediate hosts of, 6
Organic compounds, structure and insecticid
properties of, 90
Oriental sore *see under* **LEISHMANIASIS**
Ornithodorus spp.
in U.S.A., 782, 783
determination of, 783
experimentally infected with Q fever, 601
transmitting Rickettsial diseases, 448
hermsi
biology, (545)
transmitting, experimentally, *R. diaporic*
903
moubata
fertility in, (244)
toxicity, relative, of rotenone and pyrethru
in oil to, 243
transmitting, experimentally, *R. diaporic*
903
parkeri
in U.S.A., Idaho, 244
transmitting Rocky Mountain fever, 534
talaje, and relapsing fever, in Central Americ
607
tartakovskyi transmitting relapsing fever, 5
turicata
in U.S.A., 783
spirochaetal reservoir of relapsing fever, 7
venezuelensis, and relapsing fever, in Centr
America, 607
verrucosus, transmitting relapsing fever, 3
Ostertagia circumcincta, 326
Ostertagiosis, in U.S.S.R., 326
Otitis externa, desquamative
in Malta, 565
treatment, 565
Oxyuriasis *see* Enterobius infection

Paragonimiasis in Nigeria, 704
Paragonimus spp.
in cats, in China, 847
westermanii, *Didelphys virginiana* as host of, 65
Paralysis
facial, in relapsing fever, 461
popliteal, external
in Malta, 800
aetiology, 800
spastic, in Belgian Congo, 801
tick
in U.S.A., 802
caused by *Dermacentor variabilis*, 802
diagnosis, differential from
peripheral neuritis, 802
poliomyelitis, 802
Parasites
intestinal, incidence, in U.S.A., Indiana and Kentucky, 148
metazoon, of cat, in China, Kweiyang, 847
Parasitology
Clinical, Textbook of, including Laboratory Identification and Technic [BELDING] (book review), 271
Introduction to [PEARSE] (book review), 96
Medical [CULBERTSON] (book review), 873
Pasteurella
pestis
accessory growth factor, requirements of, 538
cultivation, in aerated medium, 390, 391
metabolism of, 775
and *Myco. pseudotuberculosis*, differentiation of, 139
nutrition of, 775
transmission, by fleas, experiments, 695
variability of, 47, 48
viability, 48, 49, 50
virulence of, 48, 49, 50
pseudotuberculosis, accessory growth factor, requirements of, 538
Pediculus humanus, behaviour of *S. persica* in, 55
Pellagra, (621)
in Chile, 168
Germany, 859
India, (259), 936
South Africa, 405
South-West Africa, 84
Spain, Madrid, 79, 80
U.S.A., 169, 481, 859
aetiology, 168, (259)
bodily constitution and, 80
control, 481
gastric secretion in, 79
internal secretions and, 169
of intestinal origin, 859
lesions, atypical sites of, 859
in psychiatric practice, 796
relation of excess ascorbic acid to, 259
sprue and, 860
stomach in, 79
symptoms, 405
treatment, (259), 859, 860
niacin *see* nicotinic acid
nicotinic acid, 405, 481, 936
thyroid, 936
Pemphigus foliaceus
in Brazil, 487, 488
U.S.S.R., 488
symptoms, 487, 488
treatment, 487, 488
Periarteritis nodosa and trichiniasis, relationship between, 853
Pfeifferella whitmori
isolated from case of melioidosis, 85
transmission of, 85
Pharyngostomum cordatum, in cats, in China, 848
Phenothiazine, toxicity of, 928, 929, 930, 931, 932
Phlebitis in verruga peruviana, 770
Phlebotomus spp.
of Algeria, 494, 527, 590, (871)
Anatolia, 339
Belgian Congo, biology of, 414
Colombia, 303
control of, 45
life history of, 512
naturally and experimentally infected with *L. brasiliense*, 762
minutus var. *signatipennis*, in Algeria, (871)
papatasi, life cycle of, 566
perfiliewi, in Algeria, 590
Physiology, climate and, 872
Pinta, (176)
in Mexico, 330
Usumacinta Valley, 453
Venezuela, 845
aetiology, 629
diagnosis, verification tests, 845
distribution, 629
transmission, 629
treatment, 629
Pityriasis versicolor
in Brazil, 797
treatment, 797
PLAGUE, 46–51, 139, 239–240, 306–307, 389–392, 538–539, 604–606, 695–696, 773–777, 841–843, 909, see also *Pasteurella pestis*
in Africa, South-West, 84
Argentine, 773, 774
Azores, 390
Brazil, 46, 47, 48
Canary Islands, 390
Cape Verde Islands, 390
Cuba, 390
Ecuador, 774, 909
Grenada, 390
Madagascar, 777
Mexico, 239
Morocco, 604
Palestine, 538
Peru, 306, 774
Porto Rico, 390
South Africa, Transkei, 865, 866
Trinidad, 390
Venezuela, 46, 605
Virgin Islands, 390
bacteriology, 46
cold inguinal bubo and, relationship between, 46
control
in Argentine, 773
Morocco, 604
Palestine, 538
on airplanes, 564

Plague—*cont.*
diagnosis
differential, from tularaemia, 50
intradermal reaction, 50
epidemiology, (51)
Laboratory of Hamburg Hygiene Institute, (777)
multiglandular, bacteriology of, 46
pneumonic
in South Africa, 605
Uganda, 240
decline of epidemics, 776
prophylaxis, 605
treatment, 605
prophylaxis, E.V. vaccine, 777
Summary of Recent Abstracts, 505–510
sylvatic
in U.S.A., 696, 841
control, fumigants, 306
vectors of, in North America, 841
transmission, (51), 696, 909
rôle of *Synopsyllus fonquernii* in, 842
treatment (including experimental), 50
sulphonamides, 306, 773
sulphathiazole, 306
vaccination, 604, 777
living attenuated and dead vaccine, comparative value, 391
Plants, Poisonous and Injurious, of Panama (book review), 724
Plasmodia
of Central African apes, relationship to human parasites, 438
avian, hosts of, (17)
cryptozoites, 815
low-temperature freezing of, 756
sporozoites of, 214
development of, 677
electron-optical observations on, 665
staining, 432
Plasmodium
cathemerium
exoerythrocytic schizonts, tissue distribution of, 221
schizogony, 221
falciparum
infections, parasite cycle of, 109
morphological changes in, following atebrin therapy, 292
gallinaceum, 220
hosts of, (17)
inoculation of fowls against, 362
pigment, produced by, in fowls, 440
sporozoites, development of, 221
transmission to mosquitoes, 362
knowlesi
antigen, effect of inoculation of, into infected monkeys, 755
cultivation *in vitro*, 755
plasmoquine-resistance in, 754
respiratory and carbohydrate metabolism, 674
lophurae, 222
survival *in vitro*, 675
susceptibility of *Aëdes aegypti* to, (584)
transmission by *A. quadrimaculatus*, 583
ovale, in Armenia, (110)
Plasmodium—*cont.*
relictum
exoerythrocytic schizonts, tissue distribution of, 221
gametocyte production, variation of, in canaries, 583
schwetzi
evolution of, in *A. maculipennis* var. *atroparvus*, 119
schizogony cycle of, 119
vivax, unsuccessful attempts to transmit old inoculation strain of, by Anopheles, 109
Pneumonia
associated with *Trichomonas buccalis*, 181
eosinophil, 948
treatment by sulphadiazine, 181
Poisoning
Conus textilis, 635
fish
in Porto Rico, 86
symptoms, 86
Medusa, treatment, 722
puss caterpillar, treatment, 181
Poisonous and Injurious Plants of Panama (book review), 724
Poliomyelitis, diagnosis, differential, from tick paralysis, 802
Polyneuritis, of pseudo-tabetic type
in Eritrea, 77
diagnosis, differential, from beriberi, 77
symptoms, 77
treatment, 77
Precipitin test, and production of precipitating sera, 749
Pregnancy
malaria and, 289
termination by, 431
Prickly heat
in Eritrea, 627
aetiology, 627
complications, syringadenitis suppurativa tropicalis, 862
treatment, 627, 628
Protozoa, intestinal
in Czechoslovakia, 700
Mexico, 455, 459
Natal children, 398
U.S.S.R., 313
Crimea, 911
Moscow sewage, 311
U.S.A., 53, 70, 148, 457, 697
examination of faeces for, 268
house-fly as carrier of, 698
skin diseases and, 797
study of, 52
Pseudofolliculitis, scarring, in bearded Negroes, 863
Pseudotabes, tropical
in Eritrea, 796
blood protein in, 796
and tropical neuritis, differential diagnosis, 857
Psychosis
acute, and trichiniasis, (935)
following Atepé treatment of malaria, 751
in typhus, 831
Pterygium, in Algeria, 942
Pulex irritans, in cats, in China, 847

Pyoderma ulcerosum tropicalum
in Australia, 629
symptoms, 629
treatment, 629
Pyomyositis, in East African campaign, 332
Pyrogen-free infusion fluid, 140
Pyrogenic substances in intravenous injections, tests in rabbits, 454
Q fever *see under* **TYPHUS GROUP OF FEVERS**
Quinidine, estimation of, in human plasma, 821
Quinine
ankylostomiasis and, 158
estimation of
in human plasma, 821
in urine, 880
supply problem of, 361
RABIES, 195–210, 651–662
in Austria, 205
Chile, 657, 659
Mexico, 202
U.S.A., 204
Venezuela, 208
canine, vaccine, effect of prolonged storage on, 655
diagnosis, 199
biological, 205
Negri body and, 207, 660
experimental
animals for, 653
sero-prophylaxis, 204
paralysis, postvaccinial, 205, 657, 659
pseudorabies of Aujeszky, virus
action of vapour of eucalyptus on, 198
experimental infection of chick embryo with, 206
statistics, Pasteur Institute, Paris, 1940, 203
transmission, nasal, effect of anaesthesia on, 199
treatment, statistics, Instituto Camara Pestana, 1938–1942, (662)
vaccination, 203, 657, 659
acute myelitis following, 204
paralytic accidents, 205, 657, 659
vaccine, 201
viruses
of Venezuela, 208,
action of vapour of eucalyptus on, 198
attenuation of, 196
desiccation of, 195
and equine encephalomyelitis virus, paraimmunity phenomenon between 661
fixed
comparative study of different strains of, 661
immunogenic properties of, 200
passaging of, 198
and street, differentiation, 197
path of dissemination of, in mice, 651
recovery of, from brain, 199
street, association of, with
herpes virus, 206
louping ill virus, 206
[WEBSTER] (book review), 269
Raillietina infection
in U.S.S.R., in child, (66)
in tropical countries, (614)
Rat-Bite fever
in French Equatorial Africa, 56
India, 244, 783
experimental, action of *p*-amino-phenylsulphamide in, (57)
following bite of Indian squirrel, 244
symptoms, 56
treatment
novarsenobenzol, 56
novarsenobillon, 244
Rats
commensal, 240
control, in U.S.A. cities, 842
RELAPSING FEVER AND OTHER SPIROCHAETOSES, 55–57, 243–244, 313–315, 460–462, 544–545, 607, 782–784, 916–919 *see also* Spirochaeta
in Abyssinia, 313, 632, 686
Algeria, 460, 494
Colombia, 545, 607
Costa Rica, 607
Cuba, 607
Eritrea, 857, 892, 916, 950
Guatemala, 607
Jamaica, 607
Middle East, 461
Northern Rhodesia, 607
Palestine, 55, 461
Panama, 545, 607
Spanish Honduras, 607
U.S.S.R., 314, 544
U.S.A., 545
Venezuela, 545
blood sedimentation rate, in Eritrea, 950
complications
encephalitis, 461
facial paralysis, 461
herpes febrilis, 461
ocular, 461
diagnosis, differential from
leptospirosis, 462
trench fever, 839
distribution, 782
experimental infection in G.P.I. patients, treatment, 315
simulating acute abdomen, (56)
transmission, 55
Ornithodorus spp., 782, 783
tartakovskyi, 544
verrucosus, 314
treatment
arsenic, 633
N.A.B. and sobita, 607
neosalvarsan, 917
organic arsenicals, intravenously, 314
sobita, 607
and N.A.B., 607
typhus and, 686
vitamin B deficiency and, 857
REVIEWS AND NOTICES, 96, 182–184, 340–344, 415–420, 503–504, 569–570, 641–642, 724–726, 807–808, 873–876, 951–954
Rheumatism, muscular, diagnosis, differential, from trench fever, 839
Rhinitis, in Hawaii, 635

Rhinosporidiosis
of conjunctiva
in Uganda, in child, 177
U.S.A., 492, 941
diagnosis, 177
Rhipicephalus sanguineus transmitting Rocky Mountain fever, 534
Rhodnius prolixus
integument of, relation to entry of Pyrethrum extracts, 89
naturally infected with *T. cruzi*, 680, 759
Rhopalopsyllus spp.
in Argentine, 605
Brazil, 605
Ecuador, 605
Panama, 605
Riboflavin deficiency
in Ceylon, 854
India, 936
causing keratitis
parenchymatous, 936
superficial, 491
circumcorneal injection in, 78
in disease, 559
failure of riboflavin therapy in, 559
in man, experimental, 619
ocular disturbances in, 491, 858
Rickettsiae *and see under* **TYPHUS GROUP OF FEVERS**
dermacentrophila, 238
diaporica, transmission, experimentally, by *O. moubata* and *O. hermsi*, 903
prowazeki
demonstration of, in infected embryo chicks, 895
virulence of, 381
Rickettsial diseases
of Australia, 234
diagnosis, complement fixation vaccine for antigen in, 897
Ringworm of feet, 175
Rockefeller Foundation
annual report 1941, (343)
International Health Division, annual report 1941 (book review), 340
Rocky Mountain spotted fever *see under* **TYPHUS GROUP OF FEVERS**
Roseola and leprosy, 788
Salvation Army Officers, on Missionary Service, Tropical Medical Manual for Use and Guidance of, 3rd edition [TURNER] (book review), 875
Sandflies *see* Phlebotomus
SANDFLY FEVER, 45, 305–306, 604, 694–695
in Greece, 694
India, 604
Palestine, 338, 339
U.S.S.R., 45
diagnosis
choking of optic discs, 604
differential from
Bessarabia fever, 839
dysentery, 695
influenza, 695
malaria, 695
harara and, in Palestine, 338, 339
treatment, 695
virus, cultivation, 305
Sanitation
rural, 268
small sanitary structures, design of, 799
Scarlet fever in South-West Africa, 84
Schistosoma
Bulinus contortus raymondi as interm host of, 469
destruction of, 252
Schistosoma mansoni
cercariae, lethal dose of chlorine for, 156
life history of, 155
Schistosomiasis
in Belgian Congo, 249, 250
East African campaign, 332
Germany, 63, 612
Natal, 398
South Africa, 550
Southern Rhodesia, 398
Sudan, 633
U.S.A., possibility of increase in,
Venezuela, (553)
carcinoma of pancreas and, (323)
in children, 398, 550, 553
complications, 398
control
in children, 553
in troops, 252
diagnosis
complement fixation, 63
difficulties of, 550
skin tests, 63, 471
facial appearance in, 251
prophylaxis, 251, 471
symptoms, 398
treatment
cylotropin, intravenously, 849
fouadin, intramuscularly, 849
Schistosomiasis haematobium
in Algeria, 466, 467
Canary Islands, 848
Morocco, 469
Natal, 553
Senegal, 848
control, 466, 467
diagnosis, 612
Bayer 205, 612
treatment
anthiomaline, 553
Bayer 205, sequelae, 613
fouadin, 613
tartar emetic, 613
Schistosomiasis mansoni, 703
in Brazil, 579
in children, 550
Egypt, 704
Porto Rico, 704
Venezuela, 849
experiments, 923
manifestations, anorectal and colonic, c
treatment, (613)
fouadin, 156
Sciurus spp., rat-bite fever from bite of, 2
Scorpions
of Algeria, 494
sting, 82, 485, 486
body temperature after, 486
glycaemia following, (486)

y
South Africa, Transkei, 866
outh-West Africa, 84
aemia, diagnosis, differential from trench fever, 839
precipitating, production of, precipitin test and, 749
ge
ification of, from cysts of intestinal protozoa, 311
tment of, when used as vegetable manure, 318
iidae
of Mozambique, (636)
Southern Rhodesia, 475
Venezuela, Caracas Valley, (950)
trol, 256
ium
nosum, in Belgian Congo, 214
lusaeforme, in Belgian Congo, 214
naptera
Mendoza, (240)
North America, index to literature of, (180)
ing sickness *see* **TRYPANOSOMIASIS**, human, African
pox
in Africa, South West, 84
Sudan, 633
trol, in airplanes, 564
of Algeria, 494
ngarus fasciatus, crystalline haemolysin from, 861
ja tripudians, crystalline haemolysin from, 861
u see **RAT-BITE FEVER**
anosis in Dutch East Indies, 553
r
e
in Madagascar, 862
reatment, 862
ck widow *see Latrodectus mactans*
oya" *see Latrodectus curacaoensis*
llum minus, isolation from case of rat-bite fever, 56
chaeta
panica, 461
xperimental study of two Algerian strains, 460
irulence of blood of guineapig infected with, 917
vshewi n. sp., 314
tropicalis n. sp., 607
sica
behaviour of, in *Pediculus humanus*, 55
bservations on, 55
icatae, experimental infection of *M. mexicanus* with, 314
ezuelensis, transmission of, 545
chaete, fowl, cultivation of, 917
chaetosis, North African, inborn resistance of guineapig to, (918)
trichosis
in Brazil, 174
Mozambique, 630
South Africa, Witwatersrand, 489
U.S.A., 173, 490
iology, 630

Sporotrichosis—*cont.*
diagnosis, 173, 630
differential from
streptococcic lymphangitis, 490
tuberculosis, cutaneous, 490
distribution, 630
due to *S. schencki*, 173, 174
symptoms, 489
transmission, 630
treatment, 490, 630
failure of, 173
potassium iodide, 491
sodium iodide and metaphen, 174
Sporotrichum spp., 630
SPRUE, 170, 259–262, 482–484, 621–622, 712–714, 937–938
in Brazil, 261
Germany, 937
U.S.A., 860
aetiology, 259, 261
B_2 complex in Cohn liver extract in relation to, 482
diagnosis, 712
motility of small intestine in, 937
pathogenesis, 259, 261
pellagra and, 860
and pernicious anaemia, simularities and differences between, (170)
syndrome
cutaneous changes in, 483
diagnosis, 170
treatment, resistance to, 170
treatment, 713, 860
lecithin in, 621
water absorption in, 937
Steatorrhoea and chyladenectasis, 483
Stegomyia, anti-, campaign, in South America, 537
Stomoxys, life history of, 512
Streptothrix in Greece, 864
Strongyloides stercoralis
appendicitis and, 556
infection
in Brazil, in children, 550
Dutch East Indies, Java, 326
U.S.A., 794
Kentucky, 148
treatment by phenothiazine, 702
invasion of lumen of intestine by, 326
male, found in stool, 794
Sulphonamides, medical uses of, 867
Sun
insulation against, efficiency of headgear, 502
light, and desert sores, 718
protection against, 568
stroke, treatment, 719
Surgery, tropical, in East African campaign, 331
Synopsyllus fonquernii, rôle of, in plague transmission, 842
SYPHILIS AND YAWS, 148–149, 545–546, 608, 845–847
Syphilis
in Eritrea, 949
bejel
in Euphrates valley, 608
bone lesions, radiographic study of, 608
blood sedimentation rate, in Eritrea, 949
exotic, in Annamites in Indo-China, 846

Syringadenitis suppurativa tropicalis
in Chinese, 862
treatment, 862
Taenia spp., and appendicitis, 556
Taenia taeniaformis, in cats, in China, 848
Taeniasis *see under* Tapeworm infection
Taeniorhynchus africanus, in Sudan, 602
Tapeworm infection
Diphyllobothrium infection
in U.S.S.R., 158
U.S.A., in children, 924
treatment by oleoresin of Aspidium, 925
Dipylidium caninum infection
in Czechoslovakia, 700
treatment, 430 Kl., experiments, 60
Hymenolepis
diminuta infection, in Mexico, (851)
nana infection
in Bulgaria, 790
in children, 790
var. *fraterna* infection, transmission of immunity from mother to offspring, 322
Mesocestoides variabilis infection
in U.S.A., 323
treatment by oleoresin of Aspidium, 323
taeniasis
in Belgian Congo, 251
Brazil, in children, 550
Czechoslovakia, 700
India, 925
U.S.S.R., 158
diagnosis, 474
skin tests, 474
treatment
carbon tetrachloride, 925
430 Kl., experiments, 60
hexylresorcinol, 925
phenothiazine, 702
tetrachlorethylene, 925
Tapeworms, nutrition of, 705
Temperature, and resistance to infection, 567
Tertiana perniciosa siderans
in U.S.S.R., 668
treatment, 668
Tetanus
in India, 723
treatment by magnesium sulphate, 723
Thelazia
californiensis infection, in U.S.A., 257
callipaeda, in cats, in China, 848
Thiamin deficiency, development of myocardial necrosis and absence of nerve degeneration in, 795
Thromboangiitis obliterans *see* Buerger's disease
Ticks, South African, eggs, toxin from, (567)
Tinea imbricata in India, 175
Toxocara
cati infection, treatment
ascaridol, experiments, 62
430 Kl., experiments, 60, 62
santonin, experiments, 62
mystex, in cats, in China, 848
Toxoplasma, neutralizing antibody, 636, 637
Toxoplasmosis
in U.S.A., 637, 950
congenital, in U.S.A., 950
symptoms, 951
diagnosis, complement fixation reaction, 637
treatment, experimental, 638
Trachoma.
in Algeria, 494, 942
Egypt, 492
Germany, 494
India, 494, 942
Sudan, 633
diagnosis, 942
entropion and, 942
pathology, 942
treatment
albucid, 494
sulphanilamide, 492
trichiasis and, 942
virus
antigenicity of, 941
cultivation of, 941
Weil-Felix reaction in, 492
Trench fever *see under* **TYPHUS GROUP (F FEVERS**
Triatoma, infection with *T. cruzi*
experimentally
capitata, (891)
hegneri, 891
naturally
ambigua, 590
gerstäkeri, 526, 590
hegneri, 891
heidemanni, 526, 590
infestans, 525, 681, 760, 761, 889
longipes, 590
oswaldoi, 761
protracta, 590
woodi, 590
sanguisuga, 590
sordida, 761
uhleri, 590
Triatomidae
of Chile, 889
infection, natural with *T. cruzi*, 525, 526, 5 681, 760, 761, 888, 889
Trichiasis
trachoma and, 942
treatment, 942
Trichinella antigen, active sensitization with, man, 477
Trichinella spiralis
active immunity in rats, to, 794
cultivation, in roller tube tissue cultures ,85
destruction of, (854)
effect of ultra violet radiation on, 403, 556
immunity
active, in mice, 75
passive, in rats, 77
larvae
development, in musculature, 933
in tissues other than skeletal muscles, (3
longevity and sex ratio of, 934
natural hereditary transmission of immunity 794
polysaccharide from, 328
Trichiniasis, (258), (328), (619), (935)
in Chile, (853)
Cuba, 711
Germany, 935
Mexico, 711
U.S.A., (258), (794), 853
and acute psychoses, (935)
allergy in, 935
from bear meat, (794)

Trichiniasis—*cont.*
diagnosis, 935
Bachman's reaction, (853)
complement fixation test, (329), 478
differential, from typhus, 299
skin tests, 478
electrocardiograms in, (258)
encephalitis and, (328)
hypoglycaemia in, (619)
immunological reactions in, (619)
incubation period of, (258)
and periarteritis nodosa, relationship between, 853
serological reactions in, (853)
symptoms, mental, (329)
Trichocephalus muris, immunity in white mice to superinfection with, (168)
Trichodectes spp., in cats, in China, 848
Trichomonas
in U.S.S.R., 313
blood-ingesting, in amoebiasis, 459
buccalis, associated with pneumonia, 181
hominis
commensal and parasitic forms, 911
infection
in Czechoslovakia, 700
Dutch East Indies, Java, 326
U.S.S.R., Crimea, Sebastopol, 911
U.S.A., Indiana and Kentucky, 148
Trichostrongylus colubriformis, invasion of lumen of intestine by, 326
Trichuris infection
in Belgian Congo, 251
Canary Islands, 849
Natal, in children, 398
Trichuris
serrata infection, treatment, 430 Kl., experiments, 60
trichiura
appendicitis and, 556
infection
in Brazil, 579
in children, 550
Bulgaria, 790
Czechoslovakia, 700
Dutch East Indies, Java, 326
Nigeria, in children, 497
U.S.A., 148
in children, 497, 550, 790
treatment by phenothiazine, 702
invasion of lumen of intestine by, 326
Trombicula deliensis, vector of mite-borne typhus in Australia, 234
Tropical
Diseases
in Australasia. A Handbook, 2nd edition [CILENTO] (book review), 569
prevention, (85), (495)
in troops, control, 284
in wartime, (85)
housing, 638
Medical Manual. For Use and Guidance of Salvation Army Officers on Missionary Service, 3rd edition [TURNER] (book review), 875
Medicine
American, Story of, Ambassadors in White [WILSON] (book review), 417
Tropical—*cont.*
Medicine—*cont.*
Synopsis of [MANSON-BAHR] (book review), 415
neurasthenia, 634
neuropsychiatry, 802
Nursing. A Handbook for Nurses and others going abroad [GREGG] (book review), 641
OPHTHALMOLOGY, 491–494, 941–943
surgery, in East African campaign, 331
ulcers *see* Ulcers, tropical
Tropics, health in, 806
Trypanosoma
congolense, transmitted by *G. austeni*, 887
cruzi
infecting
experimentally
T. capitata, (891)
T. hegneri, 891
naturally
Cebus apella, 680
Didelphys virginiana, 526
Rhodnius prolixus, 680, 759
Triatomidae, 525, 590, 681, 760, 761, 888, 889, 891
persistence of, in dead Triatomidae, 525
reservoir hosts of, 525, 526, 760
equiperdum, infection, treatment by stilbamidine, failure of, 377
gambiense
behaviour of, in pig, 367
transmission by *G. palpalis*, irregularities in, 368
lewisi
infection, in rat, effect of biotin deficiency on duration of, 891
reinfection, defence mechanism against, 758
Trypanosomal meningo-encephalitis, diagnosis and treatment, 120
Trypanosomes
infectivity of, during chemotherapy, 19
isolation by fractional centrifugation of blood, 758
TRYPANOSOMIASIS, 18–23, 120, 224–227, 293–295, 367–375, 524–526, 586–590, 678–682, 758–761, 885–891
animal, 885
experimental, treatment, chemotherapeutic interference between methylene blue and neoarsphenamine, 294
human, African (sleeping sickness)
in Bechuanaland, 885
Belgian Congo, 18, 224, 373
Cameroons, 18, 19, 586
French Equatorial Africa, 887
French West Africa, 524
Gambia, 224
Mozambique, 524
Northern Rhodesia, 589
Sierra Leone, 370
control
in Cameroons, 586
French West Africa, 524
diagnosis
mass, 370
stained thick blood film, 18

Trypanosomiasis—*cont.*
human, African (sleeping sickness)—*cont.*
game and, 588
Glossina and, 588
hereditary, 19
leprosy and, 149
prophylaxis
atoxyl, 586
Bayer 205, 373
reinfection, 887
survey, in Bechuanaland, 885
treatment, 887
antrypol, 370
sequelae, 371
antrypol and tryparsamide, 370
sequelae, 371
aromatic diamidines, 19, 224, 225, 370, 589, 682, 683, 684
experiments, 377
pharmacological actions of, 682, 683, 684
arsenicals, relation between chemical structure and drug-resistance among, 20
atoxyl, 586
Bayer 205, 524
chemotherapy, advances in, 179
infectivity of trypanosomes during chemotherapy, 19
mapharsen, experiments, 589
mass, 370
pentamidine, 224, 370, 589
sequelae, 225
propamidine, 370
stilbamidine, 370
tryparsamide, 370, 586
sequelae, 371
visual complications, 372
tryparsamide and antrypol, 370
sequelae, 371
human, American (Chagas's disease)
in Argentine, 22, 680
Bolivia, 525, 761, 888
Brazil, (526), 889
São Paulo, 266
Chile, 760, 761, 888
Columbia, 759
Venezuela, 375, 680
adenitis and, 227, 890
cardiac conditions in, 761
in child, 889
diagnosis, 226
complement fixation, improved antigen for, 890
Machado reaction, 760
-Guerreiro reaction, 375
xeno-, 375, 760, 889
experimental, 681
pathology, 21
histology, 227
myxoedema, study on, 21
transmission, (526), 680
treatment, Bayer 9736 (As) and 7602 (Ac) compared, 22
Summary of Recent Abstracts, 351–357
treatment by tryparsamide, amblyopia and, 679
Tsetse flies *see* Glossina
Tsutsugamushi disease *see under* **TYPHUS GROUP OF FEVERS**
Tuberculosis
in Africa, South-West, 84
Algeria, 494
Bathurst Island, 412
British Colonies, 631
Eritrea, 949
in natives, (179)
Melville Island, 412
Mexico, Usumacinta Valley, 453
Nigeria, in children, 497
South Africa, Transkei, 865
blood sedimentation in, in Eritrea, 949
control, in tropical rural areas, 155
cutaneous, diagnosis, differential from sporotrichosis, 490
diagnosis, differential, from trench fever, 839
leprosy and, 847
miliary, diagnosis, differential from typhus, 299
pseudo-, with eosinophilia, in India, 720
Tularaemia
diagnosis, differential, from plague, 50
transmission by *Haemophysalis leporis palustris*, 836
Tumours
in Chinese children, (267)
amoebic, of large intestine, treatment, 145
Typhoid fever
in Abyssinia, 632
South-West Africa, 84
diagnosis, differential, from trench fever, 839
ten-day fever simulating, in Ceylon, 239
TYPHUS GROUP OF FEVERS, 25–39, 125–137, 230–239, 297–302, 378–388, 442–450, 528–534, 591–602, 685–692, 762–769, 828–840, 892–904
in Africa, South-West, 84
Algeria, 443, 494
British Colonies, 631
Eritrea, 855, 950
French Equatorial Africa, 833
Germany, 237
Salvador, 267
South Africa, Transkei, 865
U.S.A., in vaccinated laboratory worker, 32
Bessarabia fever, 839
diagnosis, differential, from
dengue, 839
sandfly fever, 839
blood
pressure, 831
in cerebral arteries in, 234
sedimentation rate, in Eritrea, 950
Citochol reaction in, 689
classification of, 128, 230, 442
clinical aspects, 687
pathology of brain-stem, 381
control, (32)
on airplanes, 564
diagnosis, 687
differential, 30
from trench fever, 839
dry blood agglutination test, 596
dry reagent "Cracow," 133
laboratory, 230

Typhus Group of Fevers.—*cont.*
diagnosis—*cont.*
microscopical, 597
modified
micro-reaction of Castañeda and Silva, 598
Weil-Felix reaction, 600
serological, 597
dry antigen in, 600
diet and, 444
endemic typhus (including murine)
in Abyssinia, 632
Africa, West, in British troops, 384
Australia, 234,
Chile, 36, 593
Colombia, 533, 890
Egypt, 895
French Equatorial Africa, 594
French Guiana, 768
Gold Coast, 593
Honolulu, 767
Iraq, 895
Jamaica, 443
Middle East, 895
Palestine, 895
Salvador, 767
Syria, 895
U.S.A., (36), 690, 834
control, 834
distribution, 592
and epidemic typhus
combined vaccination, 128
diagnosis, differential, complement fixation test, 443
laboratory infection, 449
Rickettsiae, morphological structure of, 685
symptoms, 30, 37
transmission, 28
treatment, 29
vaccination, 128
virus, isolated from *Mus musculus musculus*, (690)
epidemic typhus (louse-borne) *and see* exanthematic typhus
in Abyssinia, 632, 686
Africa, 27
Algeria, 379, 741
Bulgaria, 126
Chile, 593
Colombia, 298
Egypt, 895
Eire, 762
Eritrea, 892
Europe, 27
France, 379
German troops, 298, 531, 831, 899
Germany, 30, 126, 379
Greece, 126
Hungary, 126
Iraq, in troops, 895
Middle East, in troops, 895
Morocco, 379
Poland, 27, 31, 126, 379
Rumania, 126
Salvador, 136
South Africa, 591
Spain, 126, 379
Tunisia, 379
U.S.S.R., 299, 379, 765

Typhus Group of Fevers—*cont.*
epidemic typhus (louse-borne)—*cont.*
U.S.S.R. in German troops, 531
Yugoslavia, 126
aetiological problem, 127
allergic reaction in, 894
blood
leucocyte picture, 595
picture in, 446
complications, 298
mental disturbances in, 831
control, 27
in Morocco, Blanc's vaccine, 379
delousing hut, in U.S.S.R., 765
vaccines, 379
diagnosis, 27, 231, 531, 686
differential from
endemic typhus, complement fixation test, 443
epidemic meningitis, 299
German measles, 299
sepsis, 299
trichiniasis, 299
tuberculosis, miliary, 299
dry-blood agglutination test, 529
Giroud intradermal test, 530
serological test, 382
Weltmann's coagulation test, 383
distribution, 592
gangrene of limbs, treatment, 445
immunization, 27
louse in relation to, (130)
malaria and, 686
and murine typhus, combined vaccination, 127
neuritis and, 899
pathological anatomy of, 444
relapsing fever and, 686
Rickettsiae
blocking of reticulo-endothelial system by, 894
in endothelial cells of bone marrow, 893
morphological structure of, 685
symptoms, 30, 298, 299, 384, 531, 686
cardiovascular, treatment, 445
mental, 831
neurological, 382
transmission, 28
treatment, 27, 30, 531, 687
chinifortan, 299
sulphonamides, 31
symptomatic, 32
trench fever and, 298
in troops, 298, 531, 831, 895, 899
vaccination, 127
vaccine, 897
in wartime, 125
Weil-Felix reaction in, 136
exanthematic *see* epidemic typhus
Exanthematic (book review), 641
Laboratory Technique of (book review), 952
experimental, 385
Proteus OX19, drying of, 898
Proteus X19, in, 134
immunity, 232
Kahn reaction in, 689
Meinicke reaction in, 689

Typhus Group of Fevers—*cont.*
mite-borne
in Australia, 234
distribution, 592
murine typhus *see* endemic typhus
non-specific luetic reactions in, 689
pathological anatomy of, 764
" pinareño " typhus, in Cuba, 127
prophylaxis, 230
Q fever
in Australia, 234, 302
U.S.A., 602
Rickettsiae
morphological structure of, 685
persistence of, in guineapig tissues after defervescence, 769
transmission, 302
experimental, 903
Haemaphysalis bispinosa, 601
Ixodes holocyclus, 388
research, 766
Rickettsia prowazeki, growth enrichment of, by intraperitoneal injections of blood, 532
Rickettsiae
biology of, 25
morphology of, 763
nomenclature of, 828
preservation by cold, 532
staining of, 297
structural phases of, 764
scrub typhus, distribution, 592
Summary of Recent Abstracts, 727–738
symptoms, 687
tick-borne
in Abyssinia, 632
Brazil, 300
Colombia, 447
Far East, 300
India, 901
South Africa, 591
U.S.A., Texas, 386
boutonneuse fever
in Eritrea, 901
distribution, 592
pseudo-, in Belgian Congo, 37
Rickettsiae, in endothelial cells of bone marrow, 893
distribution, 592
experimental, anti-rickettsial serum in, 768
Rocky Mountain fever
in U.S.A., 690, 835, 836, 837, 902
Georgia, 239
brain changes in, 690
distribution, 592
Rickettsiae, morphological structure of, 685
strains isolated, in Georgia, U.S.A., 238
symptoms, 902
transmission, 533
treatment, 902
experimental, 691
immune serum, 837
vaccine, tick-tissue, duration of potency of, 691
vectors of
in U.S.A., 533
Amblyomma americanum, 534, 835
cayennense, 534

Typhus Group of Fevers—*cont.*
tick-borne—*cont.*
Rocky Mountain fever—*cont.*
vectors of—*cont.*
Haemaphysalis leporis-palustris, 836
Ornithodorus parkeri, experiments, 533
symptoms, 300, 591
transmission, experiments, 386, 387
virus experiments, 386, 387
treatment, 230, 687
adrenal cortical extract, 237
atebrin and calcium. 237
convalescent blood, 236
hot bath, effect of, on blood pressure, 831
trench fever
in Belgium, 301
France, 301
Germany, 38
Italy, 301
Poland, 301
in German troops, 903
Rumania, 301
U.S.S.R., 301
in German troops, 446, 838, 903
aetiology, 136
diagnosis, 387
differential from
encephalitis, 839
influenza, 839
malaria, 839
relapsing fever, 839
rheumatism, muscular, 839
septicaemia, 839
tuberculosis, 839
typhoid fever, 839
typhus, 839
and epidemic typhus, 298
symptoms, 39, 838, 903
treatment, 301, 447
atebrin, 39
neosalvarsan, 39
in troops, 446, 838, 903
types of, in German troops, 446
variations in, 136
tsutsugamushi disease
in Australia, 234
British Colonies, 631
distribution, 592
Ukrainian fever, 692
in U.S.S.R., in German troops, 903
symptoms, 903
in vaccinated laboratory worker, 32
vaccination
experiments, 34, 35, 36, 129
Weigl's vaccine, 596
virus, detection of, in reconvalescents, 893
vitamin A and, 855
Wassermann reaction in, 689
Weil-Felix reaction, 130, 236, 528, 596, 59
686, 687, 688
in convalescence, 893
in dogs, in Mexico City, (301)
rise and fall of titre in, 132
Ukrainian fever *see under* **TYPHUS GROUP FEVERS**
Ulcers
chronic, treatment by vitamin C, 333
mycotic, of cornea, in Argentine, treatme
715

Ulcers—*cont.*
phagedaenic, treatment by sterile ox-bile, 332
tropical
in Africa, 868
Assam, 717
Brazil, 546
East African campaign, 331
Eritrea, 869
Nyasaland, 716
Tanganyika, native tribe, 868
bone, radiological appearances of, 868
treatment, 717, 868, 946
surgical, 869
Uncinaria spp., in cats, in China, 848
Undulant fever
in East African campaign, 332
South-West Africa, 84
West Africa, in British troops, 385
Urticaria
malaria and, 359
solar, 94
Venereal diseases
in Abyssinia, 633
Africa, South-West, 84
Algeria, 494
Bathurst Island, 412
British Colonies, 631
Melville Island, 412
South Africa, Transkei, 865
Sudan, 633
VENOMS AND ANTIVENENES, 81–84, 172–173, 263–266, 408–410, 485–487, 560–561, 624–626, 861–862, 938–940
Venoms
scorpion, 485, 486
in Brazil, 82
snake, 81
of India, pharmacology of, 172
Acanthopis antarcticus, 264
action of, on proteolytic system in blood, 81
Ancistrodon
blomhoffi ussuriensis, haemostatic properties of, (173)
halys caragamus, pharmacology of, (173)
Australian, treatment, 263
Bitis
gabonica, 626
lachesis, 626
blood changes by, 408
Bothrops
alternata, coagulant properties of, 408
atrox, haemolytic and coagulant properties of, 408, 560
erythromelas, haemolytic and coagulant properties of, 408
jararaca, coagulant properties of, 408
Crotalus terrificus, haemolytic and coagulant properties of, 408
Demansia textilis, 263
Dendroaspis viridis, 626
Denisonia superba, 263
Elaps lemniscatus, haemolytic properties of, 408
Haemachatus haemachatus, 626
Lachesis muta, haemolytic and coagulant properties of, 408
Naja
haie, 626
Venoms—*cont.*
snake—*cont.*
naja, 265
nigricollis, eye lesions due to, 81
nivea, 626
tripudians, haemolytic properties of, 408
Notechis scutatus, 263
Oxyuranus scutellatus, 264
prevention of, 625
Pseudechis porphyriacus, 263
therapeutic use, 172
tissue injury by trypsin, 409
treatment, 625
Vipera spp., 560
berus berus, toxicity of serum, 624
ursini renardi, pharmacological properties of, 81
spider,
black widow, 83, 266, 862, 939
Coya, 486
Verruga peruviana *see under* **BARTONELLOSIS**
Vibrios *see under* **CHOLERA**
Vitamin
deficiency
in Ceylon, 854
India, Mysore, 711
metabolism of West African natives, 13
Vitamin A
blood-content of, relation to deficiency of, 855
deficiency, in Eritrea, 854, 855, 856
relationship of, to resistance to *Nippostrongylus muris*, 793
typhus and, 855
Vitamin B deficiency
in Nigeria, 480
South Africa, (796)
relapsing fever and, 857
Vitamin B_1
beriberi and, 404
deficiency
induced, in man, 558
in infants, in India, 935
Vitamin C, malaria and, 13
Vomiting sickness, in Jamaica, 945
Wassermann reaction
malaria and, 669
in typhus, 689
Weights and heights of Cantonese adult males, (96)
Weil-Felix reaction
in trachoma, 492
typhus, 130, 236, 528, 596, 597, 686, 687, 688
Weil's disease *see* **LEPTOSPIROSIS**
Whooping cough, in Algeria, 494
Wolhynian disease *see* **TYPHUS GROUP OF FEVERS**, trench fever
Wuchereria
bancrofti, transmission mechanism of, by *Culex fatigans*, 71
perstans, in Belgian Congo, 214
Xenopsylla cheopis
in China, in cats, 847
Venezuela, Caracas, 605
YAWS AND SYPHILIS, 148–149, 545–546, 608, 845–847, 919–921
Yaws
in Abyssinia, 632, 920
Africa, South-West, 84

Yaws—*cont.*
in Bathurst Island, 412
Brazil, 546
Liberia, 545
Melville Island, 412
Sierra Leone, 919
Sudan, 602
Venezuela, 546, 845
bone, X-ray appearances of, 919
control, in Brazil, 546
and dermatitis, nodular and ulcerative, relationship between, 148
diagnosis, verifications test, 845
treatment by spirobismol, 545
YELLOW FEVER, 39–44, 303–304, 388–389, 451–453, 534–538, 602–603, 692–694, 771–773, 840, 907–908
Yellow fever, (603)
in Brazil, 694
Cameroons, 907
Colombia, 694
French Equatorial Africa, 907
Gabon, 907
Mexico, 388, 453
Nigeria, 303
South America, 536
Spanish Guinea, 603
Sudan, 602
Uganda, 304
diagnosis, differential, from leptospirosis, 462
encephalitis, immunity, experiments, 772
Yellow Fever—*cont.*
immunization, 692
with 17D strain, duration of, 451
inoculation of airplane passengers, 564
jungle
in Bolivia, 536
Brazil, 536
Colombia, 536
Peru, 536
Venezuela, 536
relation of Haemagogus spp. to, 537
leptospirosis and, pathological differences between, 304
protection tests
in Nigeria, 303
Spanish Guinea, 603
intraperitoneal modified, 534
Summary of Recent Abstracts, 97–101
vaccination, 537, 907
encephalitis following, 44
jaundice following, 40, 41
vaccine, aqueous-base, 692
virus
experiments, 772
neurotropic
action of certain surface-acting substances on, 389
French strain, variation in tropism of, 40
persistence of, in brains of cerebrally immunized monkeys, 840
reaction of *Cercopithecus aethrops centralis* to, 771
susceptibility of poikilothermal animals to, 694

INDEX OF COUNTRIES.

EUROPE

Albania
Anopheles of, 286
malaria, 286, 287
rabies, 205
Balkans
leprosy, 59
Belgium
Anopheles of, 214
malaria, 214
trench fever, 301
Bulgaria
ascariasis, 790
Enterobius infection, 790
Hymenolepis infection, 790
leishmaniasis, canine, 121
leprosy, 59
myiasis of tongue, 93
Trichuris infection, 790
typhus, 126
Crete
leprosy, 59
Croatia
malaria, masked, 291
Czechoslovakia
ascariasis, 700
Blastocystis hominis infection, 700
Czechoslovakia—*cont.*
Chilomastix mesnili infection, 700
Dientamoeba fragilis infection, 700
Dipylidium caninum infection, 700
dysentery, bacillary, 700
Endolimax nana infection, 700
Enterobius infection, 700
Giardia intestinalis infection, 700
Iodamoeba bütschlii infection, 700
malaria, 217
taeniasis, 700
Trichomonas intestinalis infection, 700
Trichuris trichiura infection, 700
Eire
ascariasis, 852
typhus, 762
Europe
typhus, 27
France
Anopheles hyrcanus, (667)
maculipennis, (359)
favism, 722
trench fever, 301
typhus, 379
Germany
amoebiasis, 606, 781, 913
Anopheles maculipennis races, 7
ascariasis, 792

Germany—*cont.*
cholera, Hamburg epidemic of 1892, (140)
dysentery, bacillary, 143
malaria, (108), 669
pellagra, 859
plague laboratory of Hygiene Institute at Hamburg, (777)
schistosomiasis, 63, 612
sprue, 937
trachoma, 494
trichiniasis, 935
typhus, 30, 126, 237, 379
in troops, 298, 831, 899
Great Britain
Clonorchis sinensis infection, 157
dysentery, bacillary, 779
hydatid disease, in Wales, 850
malaria, 579
Greece
actinomycosis, 864
Anopheles of, 429
blastomycosis, 864
leprosy, 59
malaria, 292, 427
sandfly fever, 694
Streptothrix infection, 864
typhus, 126
Hungary
typhus, 126
Italy
Anopheles hyrcanus var. *pseudopictus*, 580
Filaria conjunctivae, 927
trench fever, 301
Lithuania
leprosy, 245
Malta
external popliteal paralysis, 800
leprosy, in troops, (248)
otitis externa, desquamative, 565
Mediterranean countries
kala azar, 376
Montenegro
leprosy, 59
Poland
dysentery, giardial, 147
trench fever, 301, 903
typhus, 27, 31, 126, 379
Portugal
leprosy, 60
malaria, 818
Epidemiology of, (book review), 951
Rumania
leprosy, 59
trench fever, 301
typhus, 126
Spain
cysticercosis, cerebral, 65
kala azar, 23, 120, 375
leprosy, 60
malaria, 751
pellagra, 79, 80
typhus, 126, 379
Turkey
Anopheles of, 816
sergenti, 816
oriental sore, 125
U.S.S.R.
amoebiasis, 312, 313
U.S.S.R.—*cont.*
Anopheles of, 824
control, 437, 519, 520, 824
Bessarabia fever, 839
Chilomastix infection, 313, 911
Diphyllobothrium infection, 158
dysentery, 143, 459
bacillary
in German troops, 393
balantidial, 313, 911
Endolimax infection, 313, 911
Entamoeba coli, 313
histolytica, 911
Giardia intestinalis infection, 313, 911
in children, 313
infectious diseases, 716
Iodamoeba bütschlii infection, 313, 911
kala azar, 295
leishmaniasis, 24, 441
leprosy, 701
Loa extraocularis infection, 326
malaria, 431, 433, 436, 668
control, (14)
Recent Work on (special article), 345–351
myiasis, 871
opisthorchiasis, 613
oriental sore, 296, 297
ostertagiosis, 326
pemphigus foliaceus, 488
Raillietina infection, in child, (66)
relapsing fever, 314, 544
sandfly fever, 45
taeniasis, 158
tertiana perniciosa siderans, 668
trench fever, 301
in German troops, 446, 838, 903
Trichomonas infection, 313, 911
typhus, 299, 379, 765
in German troops, 531
Ukrainian fever, 692, 903
Yugoslavia
Anopheles of, 429
leprosy, 59
malaria, 429
typhus, 126

ASIA

Anatolia
harara, 339
Phlebotomus spp., 339
Armenia
Plasmodium ovale (110)
Assam
filarial lymphangitis, 160
filariasis, 160
mosquitoes, 160
ulcers, tropical, 717
Burma
leprosy, 153
Ceylon
infant mortality, 854
keratomalacia, 854
labour conditions, 866
malaria, 746
nutrition, 854
ten-day fever simulating typhoid fever, 239
China
ankylostomiasis, 851
Anopheles of, 666

China—*cont.*
beriberi, 404
blackwater fever, 827
deficiency diseases, 404
fly population of, 871
heights and weights of Cantonese adult males, (96)
kala azar, 229
leishmaniasis, of lymph glands, 228
malaria, 666
parasites of cat, 847
tumours, in children, (267)
Dutch East Indies
ankylostomiasis, 326
ascariasis, 326
echinostomiasis, 326
leprosy, 152
malaria, 107, 750
Necator infection, 326
sparganosis, 553
Strongyloides infection, 326
Trichostrongylus infection, 326
Trichuris infection, 326
Eastern Mediterranean countries,
cirrhosis, hepato-lienal, 333
Far East
tick fever, 300
Hong Kong
cholera, 843
India
adenitis and keratoconjunctivitis, 492
age, determination of, in Bengali girls, 181
amoebiasis, 606
anaemia, hookworm, 68, 400, 406
ankylostomiasis, 68, 170
Anopheles of, 8, 114
of Nilgiris, 106
blackwater fever, 677
cataract, 494
cholera, 139, 140, 454
cirrhosis, hepatic, 947
corneal leukomata, 492
dacryocystitis, 494
dracontiasis, 256
drug addiction, 87
eosinophile-lung, 720
glaucoma, 494
kala azar, 121
keratitis, superficial, 491
keratoconjunctivitis, 494
and adenitis, 492
keratomalacia, 494
leishmaniasis, dermal, 121
leprosy, 57, 397, 546, 701, 784
research department, Calcutta School of Tropical Medicine, annual report 1941, (546)
leptospirosis, 783
malaria, 105, 112, 116, 170, 219, 663, 668, 673, 749, 753
control, 752
noma, 947
pellagra, 936
rat-bite fever, 244, 783
riboflavin deficiency, 936
sandfly fever, 604
snake venoms, 172
taeniasis, 925
tetanus, 723
India—*cont.*
Tinea imbricata, 175
trachoma, 494, 942
tropical eosinophilia, 407
typhus, tick-borne, 901
vitamin deficiency, 711
B_1 deficiency, in infants, 935
Indo-China
melioidosis, 85
syphilis, exotic, in Annamites, 846
Iraq
bejel, in Euphrates River valley, 608
typhus, 895
Lebanon
Anopheles of, 110
malaria, 110
Malaya
labour conditions, 866
leprosy, 153
Palestine
amoebiasis, 213
Anopheles of, 287
favism, (722)
filariasis, in negro troops, 926
harara, 338, 339
plague, 538
relapsing fever, 55, 461
sandfly fever, 338, 339
typhus, 895
Philippine Islands
Anopheline Mosquitoes of, and of Northern Half of Western Hemisphere (book review), 954
Enterobius infection, 617
leprosy, 464, 465
malaria, 13
Syria
ankylostomiasis, 614
Anopheles of, 110
harara, 339
malaria, 110
typhus, 895

AFRICA

Abyssinia
dysentery, bacillary, 632
kala azar, 229, 632
leech infection, in child, 869
malaria, 632, 686, 744
relapsing fever, 313, 632, 686
typhoid fever, 632
typhus, 632, 686
venereal diseases, 632
yaws, 632, 920
Africa
Food, Primer for Use in Schools, Colleges, Welfare Centres, etc., in (book review), 953
leptospirosis, 462
typhus, 27
ulcers, tropical, 868
Africa, Central
calcaemia, in natives, (267)
Africa, East
Anopheles control, 671
blackwater fever, 677
crab yaws, 332
desert sore, 499

Africa, East—*cont.*
dracontiasis, 332
filariasis, 332, 616
kala azar, 332
malaria, 332
pyomyositis, 332
schistosomiasis, 332
ulcers, tropical, 332
undulant fever, 332
Africa, French Equatorial
beriberi, (168)
goitre, 948
leprosy, 155
rat-bite fever, 56
trypanosomiasis, 887
typhus, 594, 833
yellow fever, 907
Africa, French West
trypanosomiasis, 524
Africa, Spanish West
leprosy, 149
Africa, South-West
amoebiasis, 84
ankylostomiasis, 84
anthrax, 84
dysentery, bacillary, 84
helminthiasis, 84
influenza, 84
leprosy, 84
malaria, 84
meningitis, cerebrospinal, 84
pellagra, 84
plague, 84
scarlet fever, 84
scurvy, 84
smallpox, 84
tuberculosis, 84
typhoid fever, 84
typhus, 84
undulant fever, 84
venereal diseases, 84
yaws, 84
Africa, West
airlines, medical service for, 943
amoebiasis, 945
dermatoses, 945
dysentery, bacillary, in British troops, 385
helminthiasis, 702
malaria, in British troops, 385
respiratory diseases, 945
typhus, in British troops, 384
undulant fever, in British troops, 385
vitamin metabolism of natives, 13
Algeria
Anopheles of, 494, 740
ascariasis, 494
cerebrospinal fever, 494
Enterobius infection, 494
infant mortality, 494
kala azar, 892
malaria, 105, 430, 738, 739, 740, 741
measles, 494
myiasis, ocular, (566)
oriental sore, 494, 527, 528, 590
Phlebotomus spp., 527, 590
pterygium, 942
relapsing fever, 460, 494
schistosomiasis, 466, 467
trachoma, 494, 942
Algeria—*cont.*
tuberculosis, 494
typhus, 379, 443, 494, 741
venereal diseases, 494
Angola
Anopheles of, 744
malaria, 744
Bechuanaland
trypanosomiasis, 885
Belgian Congo
ankylostomiasis, 251
Anopheles of, 7, 357
ascariasis, 251
boutonneuse fever, pseudo-, 37
child welfare work at Mayombe, 177, 178
Culicoides grahami, 214
demographic studies, in Nepoko, 495
depigmentation and oedema syndrome, 406
dermatitis, 148, (176)
haematology, 262
leprosy, 316, 397
loa loa, 214
malaria, 102, 103, 104, 210, 211, 212, 213, 218, 357, 743
molluscs, 249
Necator infection, 251
onchocerciasis, 162, 167, 214, 327
Phlebotomus, spp., biology of, 414
schistosomiasis, 249, 250
Simulium damnosum, 214
medusaeforme, 214
spastic paralysis, 801
taeniasis, 251
Trichuris infection, 251
trypanosomiasis, 18, 224, 373
Wuchereria perstans, 214
British Somaliland
beriberi, in native troops, 856
Cameroons
filariasis, 160
Jersild syndrome, 803
loa loa, in American missionaries, 926
myositis, tropica, 719
trypanosomiasis, 18, 19, 586
yellow fever, 907
Canary Islands
ascariasis, 849
Enterobius infection, 849
helminthiasis, 849
plague, 390
schistosomiasis, 848
Trichuris infection, 849
Cape Verde Island
epididymofuniculitis, filarial, 709
plague, 290
Egypt
fly control, 91
myiasis, ocular, (566)
schistosomiasis, 704
trachoma, 492
typhus, 895
Eritrea
Anopheles of, (215)
beriberi, 857
boutonneuse fever, 901
Buerger's disease, 135
dengue, 908
eye diseases, (494)
hemeralopia, 856

Eritrea—*cont.*
malaria, 880
Naja nigricollis venom, 81
neuritis, tropical, 857
oriental sore, 124
polyneuritis of pseudo-tabetic type, 77
prickly heat, 627
pseudo-tabes, tropical, 796
relapsing fever, 857, 892, 916, 950
syphilis, 950
tuberculosis, 950
in natives, 179
typhus, 855, 892, 950
ulcers, tropical, 869
vitamin deficiency, 854, 855, 856
Fernando Po
internal diseases, 179
Gabon
filarial lymphangitis, 72
yellow fever, 907
Gambia
trypanosomiasis, 224
Gold Coast
blackwater fever, 585
myositis tropica, 719
onchocerciasis, 73
typhus, 593
Kenya
Glossina of, 293, 294
control, 678
habaswein itch, 562
leishmaniasis, 230
leprosy, 154
malaria, in turkeys, 363
onchocerciasis, 73
Liberia
yaws, 545
Madagascar
plague, 777
spider-bite, 862
Mauritius
labour conditions, 866
Middle East
dysentery, bacillary, 241, 307, 308, 394, 395, 540, 777
relapsing fever, 461
typhus, 895
Morocco
ankylostomiasis, 708
health services, (563)
malaria, 742
service, annual report 1939, (284)
plague, 604
schistosomiasis, 469
typhus, 379
Mozambique
Anopheles of, 110, 111, 112
Ceratopogonidae of, (636)
entomology, (180)
malaria, 110
Simuliidae of, (636)
sporotrichosis, 630
trypanosomiasis, 524
Natal
amoebiasis, 398
ankylostomiasis, 398
ascariasis, 398
schistosomiasis, 398, 553
Trichuris infection, 398
Nigeria
alimentary diseases, 497
amoebiasis, 497
anaemia, 497
ankylostomiasis, 497
Armillifer (*Porocephalus*) *armillatus* infection, 415
ascariasis, 497
avitaminosis, 497
blackwater fever, 522
bronchopneumonia, 496
cirrhosis, biliary, 497
colitis, 497
diphtheria, 497
infant mortality, 496
leprosy, 701
malaria, 497
meningitis, 497
myositis tropica, 719
paragonimiasis, 704
respiratory diseases, in children, 496
Trichuris infection, 497
tuberculosis, 497
vitamin B deficiency, 480
yellow fever, 303
Northern Rhodesia
relapsing fever, 607
trypanosomiasis, 589
Nyasaland
ulcers, tropical, 716
Rhodesia
Glossina control, 588
Senegal
onchocerciasis, 848
schistosomiasis, 848
Sierra Leone
trypanosomiasis, 370
yaws, 919
Southern Rhodesia
schistosomiasis, 398
Simuliidae of, 475
Spanish Guinea
yellow fever, 603
Sudan
Anopheles gambiae, 288
dracontiasis, 633
dysentery, bacillary, 633
kala azar, 23, 122, 684
leprosy, (609), 633
malaria, 633
measles, 633
schistosomiasis, 633
smallpox, 633
trachoma, 633
venereal diseases, 633
yellow fever, 602
Tanganyika Territory
Culicines of, 39
health of Sandawe Tribe, 867
lathyrism, 869
malaria, 879
meningococcal septicaemia simulating, 2
ulcers, tropical, in native tribes, 868
Tunisia
coccidiosis, 805
typhus, 379
Uganda
anaemia, deficiency, 329, 484
Anopheles, control, 672

Uganda—*cont.*
plague, pneumonic, 240
rhinosporidiosis, 177
yellow fever, 304
Union of South Africa
chromoblastomycosis, 630
eye infestation by nematode, 554
histoplasmosis, 176
infant mortality, 865
leprosy, 611
malnutrition, 865
oedema, nutritional, 865
pellagra, 405, 865
plague, 865
pneumonic, 605
schistosomiasis, 550
scurvy, 865
sporotrichosis, 489
tuberculosis, 865
typhus, 591, 865
venereal diseases, 865
vitamin B deficiency, (796)

AMERICA, NORTH

America, North
Siphonaptera, index to literature of, (180)
Azores
plague, 390
Bathurst Island
ankylostomiasis, 412
bila bila, 412
leprosy, 412
medical conditions, 412
respiratory diseases, 412
tuberculosis, 412
venereal diseases, 412
yaws, 412
Canada
Enterobius infection, 328, 617, 710
Melville Island
anklyostomiasis, 412
bila bila, 412
leprosy, 412
medical conditions, 412
respiratory diseases, 412
tuberculosis, 412
venereal diseases, 412
yaws, 412
United States
ainhum, 85
amoebiasis, 53, 70, 697
of penis, 457
amoebic liver abscess, 243, 915
anaemia, sickle-cell, 407, 623
aniacinosis, 169
ankylostomiasis, 70, 324
control, in Georgia, 324
Anopheles, of Georgia, 215
appendicitis, helminths and, 555, 556, 927
blastomycosis, 562, 940
Cochliomyia americana infection, (724)
colitis, amoebic and bacillary, 697
Diphyllobothrium latum infection, 924
dysentery, bacillary, 52, 142, 396, 543, 780
Enterobius infection, 617, 618, 927
appendix and, 555, 927
fleas of, 841
United States—*cont.*
granuloma
coccidioidal, 411
venereum, of eyelid, 493
helminths, appendicitis and, 555, 556, 927
Hippelates, 339
histoplasmosis, 330, 798
of knee, 177
intestinal protozoa, 53, 70, 148, 457, 697
keratoconjunctivitis, 493
Latrodectus mactans, 83
leprosy, 609, 610, 847
leptospirosis, 304, 919
malaria, 109, 215, 290, 436, 579, 664, 665, 673, 819
control, in Tennessee, 15
in navy, 822
therapeutic, 11
in troops, 283
Mesocestoides variabilis infection, 323
onchocerciasis, 709
oriental sore, 377
Ornithodorus spp., 782
pellagra, 169, 481, 859
plague, sylvatic, 696, 841
Q fever, 602
rabies, 204
rat control, in cities, 842
relapsing fever, 545
rhinosporidiosis of conjunctiva, 492, 941
Rocky Mountain fever, 239, 690, 835, 836, 837, 902
sporotrichosis, 173, 490
sprue, 860
Strongyloides stercoralis infection, 794
Thelazia californiensis infection, 258
tick paralysis, 802
toxoplasmosis, 637
congenital, 950
trichiniasis, (258), (794), 853
typhus, (36), 690, 834
tick-borne, 386
in vaccinated workers, 32

AMERICA, CENTRAL

Costa Rica
relapsing fever, 607
Guatemala
Anopheles of, 292, (878)
malaria, 292
relapsing fever, 607
Mexico
amoebiasis, 455, 459
ankylostomiasis, 453
Anopheles of, (878)
blastomycosis, 410
dysentery,
bacillary, 460
balantidial, (782)
Entamoeba gingivalis infection, 455
Enterobius infection, 617
Hymenolepis diminuta infection, (851)
intestinal protozoa, 455, 459
leishmaniasis, 441, 453
leprosy, 609
malaria, 453
Mansonella ozzardi, 476

Mexico—*cont.*
melanosis of Riehl, 482
mycetoma, 489
pinta, 330, 453
plague, 239
rabies, 202
trichiniasis, 711
tuberculosis, 453
yellow fever, 388, 453
Nicaragua
Anopheles of, 359
Panama
dermatitis venenata, 863
Mansonella ozzardi, 476
Plants, Poisonous and Injurious of (book review), 724
relapsing fever, 545, 607
Salvador
Anopheles of, 108
malaria, 107, 267,
mosquitoes of, 108
nutritional oedema in children, 712
typhus, 136, 267, 767
Spanish Honduras
relapsing fever, 607

WEST INDIES

Antigua
leprosy, 463
Barbados
leprosy, 463
Cuba
anaemia, sickle-cell, 714
Anopheles, 745
chromoblastomycosis, 174
malaria, 431, 745
mycetoma, 630
plague, 390
relapsing fever, 607
trichiniasis, 711
typhus, 127
Dominica
Mansonella ozzardi, 476
Grenada
Anopheles of, (512)
plague, 390
Jamaica
leprosy, 462
relapsing fever, 607
typhus, 443
vomiting sickness, 945
Martinique
epididymofuniculitis, filarial, 709
Porto Rico
Anopheles of, 754
fish poisoning, 86
Giardia infections, 459
malaria, 753
plague, 390
schistosomiasis, 704
St. Kitts and Nevis
leprosy, 463
St. Lucia
leprosy, 463
Mansonella ozzardi, 476
Trinidad
leprosy, 463
plague, 390
Virgin Islands
plague, 390
West Indies
public health, 632

AMERICA, SOUTH

America, South
yellow fever, 536
Argentine
amoebic lung and liver abscess, 698
Anopheles of, 745
Chagas's disease, 22, 680
chromoblastomycosis, 491
Enterobius infection, appendiceal, 402
kala azar, 892
leishmaniasis, muco-cutaneous, 125
malaria, 219, 745
Mansonella ozzardi, 476
mycotic ulcer of cornea, 715
plague, 773, 774
Bolivia
Chagas's disease, 761, 888
kala azar, 892
trypanosomiasis, 525
yellow fever, jungle, 536
Brazil
Aëdes aegypti control, 670
anaemia, sickle-cell, 171
ankylostomiasis, 266
Anopheles, 513, 514, 515, 746
gambiae, 522, 580, 581, 670, 815
ascariasis, 579
in children, 550
Chagas's disease, 266, (526), 889
Enterobius infection, in children, 550
Health Department of, report for 1941 (book review), 182
helminthiasis, in children, (63)
kala azar, 892
leishmaniasis, 266, 546
leprosy, 150, 921
rat, (922)
malaria, 266, 510, 513, 514, 515, 518, 546, 579, 670, 746, 824
Necator infection, 579
in children, 550
pemphigus foliaceus, 487, 488
pityriasis versicolor, 797
plague, 46, 47, 48
schistosomiasis, 579
in children, 550
scorpion stings, 82
sporotrichosis, 174
sprue, 261
Strongyloides infection, in children, 550
taeniasis, in children, 550
Trichuris infection, 579
in children, 550
typhus, 300
ulcers, tropical, 546
yaws, 546
yellow fever, 694
jungle, 536
British Guiana
Anopheles of, 513
leishmaniasis, 442
malaria, 512

Chile
Chagas's disease, 760, 761, 888
Latrodectus mactans, 83
pellagra, 168
rabies, 657, 659
trichiniasis, (853)
typhus, 36, 593
Colombia
bartonellosis, 904
Chagas's disease, 759
Dermatobia, 804
Latrodectus curacaoensis, 486
leishmaniasis, 442
leprosy, 248, 549
Mansonella ozzardi, 476
Phlebotomus, spp., 303
relapsing fever, 545, 607
typhus, 298, 447, 533, 890
verruga peruviana, 302
yellow fever, 694
jungle, 536
Dutch Guiana
Mansonella ozzardi, 476
Ecuador
plague, 774, 909
French Guiana
typhus, 768
Peru
bartonellosis, 904
Carrión's disease, 905
plague, 306, 774
yellow fever, jungle, 536
Venezuela
amoebiasis, 51
Chagas's disease, 375, 680
chromoblastomycosis, nasal and laryngeal, 715
dysentery, bacillary, 51
kala azar, 891
leishmaniasis, 25
Loeffler's syndrome, 721
pinta, 845
plague, 46, 605
rabies, 208
relapsing fever, 545
schistosomiasis, (553), 849
Venezuela—*cont.*
Simuliidae of Caracas Valley, (950)
yaws, 546, 845
yellow fever, jungle, 536

AUSTRALASIA

Australia
diseases communicable from animals, (499)
dysentery, bacillary, 779
Enterobius infection, 932
helminthiasis, (923)
pyoderma ulcerosum tropicalum, 629
Q fever, 234, 302
typhus fever, 234

PACIFIC ISLANDS

Cook Islands
leprosy, 315
Fiji
leprosy, 315, 700
Gilbert Islands
leprosy, 315
Guam
infectious diseases, 716
Hawaii
asthma, 635
dermatitis, due to *Grevillea banksii*, 175
rhinitis, 635
typhus, 767
Japanese Mandated Islands
infectious diseases, 716
Melanesia
malaria, 816
Micronesian Islands
dermatitis, 870
insects of, 870
Pacific Islands
malaria, 107
Palau Islands
dengue, 870
Papua
blackwater fever, 884

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